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AMERICAN COLLEGE OF CHEST PHYSICIANS

René Laënnec

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3. Krasno et al. (1949), J. Allergy, 20:111, March

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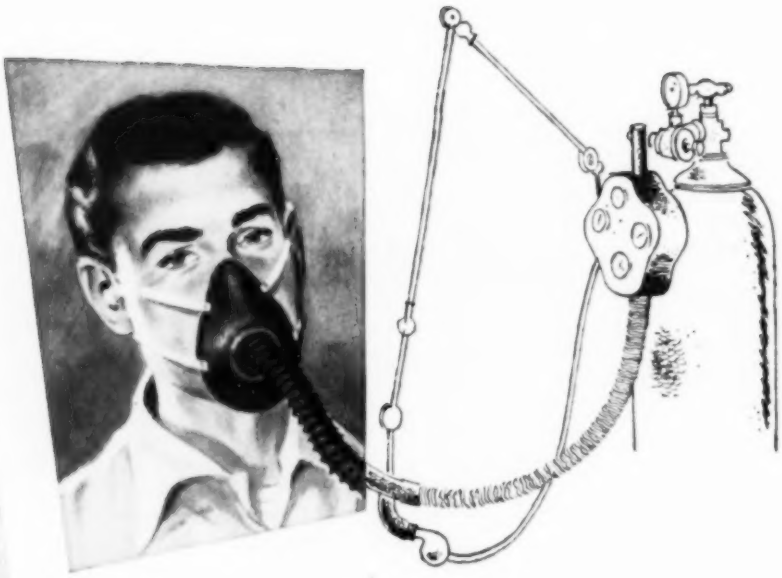

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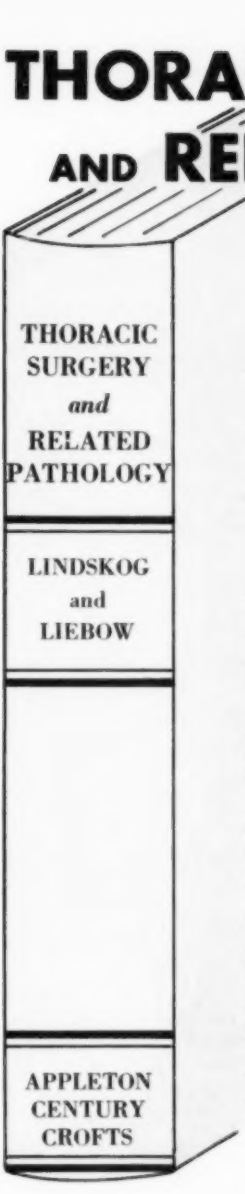

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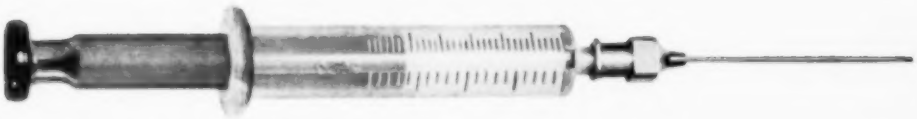

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DISEASES *of the* CHEST

VOLUME XXIV SEPTEMBER 1953 NUMBER 3

The Initial Period of Artificial Pneumothorax

The Effect of Para-Aminosalicylic Acid and Dihydrostreptomycin on the Frequency of Pleural Effusion and on Pulmonary Function*

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Gothenburg, Sweden

Introduction

Since specific antimicrobial agents produced their revolutionary effects on the treatment of pulmonary tuberculosis the need has arisen to clarify the extent to which the older methods of treatment should continue to be used. Although it is now generally agreed that the new drugs cannot supplant old-established procedures, some uncertainty still prevails regarding the indications for these measures and practice varies widely in different countries. In earlier papers from Soderby Hospital[1,2] the indications for, and the results of surgical collapse procedures in combination with specific medication were reported. There was a great increase in the number of interventions and the results were very satisfactory. In the present paper the effect of antimicrobial agents on the initial period of artificial pneumothorax therapy is investigated. Comparison is made with pneumothorax patients who did not receive such medication. In order to ascertain the trend of the results as quickly as possible, the period of observation was limited in all cases to six months after pneumothorax induction.

For a short introductory period the favorable effect of antimicrobial medication led us to consider artificial pneumothorax superfluous in early cases of tuberculosis. And also during the time covered by the study there was introduced a method for the treatment of pleural effusion which appears to have effected a significant improvement in the functional results of pneumothorax therapy.[3] When, therefore, one compares cases in which neither antimicrobial agents nor this later adjuvant were given with those in which both were systematically administered, the comparison will be between an older method and a newer which is different in many respects, but in which specific medication is the most important addition.

*From Soderby Hospital, Sweden, Department II.

This investigation has been carried out with the help of a grant from the Swedish National Association against Tuberculosis.

Material

The study comprises all the cases in which artificial pneumothorax was induced and maintained for a shorter or longer period from January 1, 1948 to October 1, 1951 with the exception of the first six months of 1949, the period when para-aminosalicylic acid (PAS) began to be given in preparation for pneumothorax therapy. This period is excluded because, the drug not being available in adequate quantity to treat all patients, only those with the poorest prognosis received it.

The clinical material is divided into three groups, as shown in Table I. That from the first period of the study, designated the "no chemotherapy group" comprised 105 consecutively-treated patients (116 pneumothoraces). Of these patients 10 had received PAS, but were retained in this group so that its consecutive composition might not be disturbed. In the second part of the investigation 82 patients (82 pneumothoraces) were treated and of these 75 received PAS. The other seven, however, are included in this "PAS group" for the reason given above. The final group consisted of 79 patients (85 pneumothoraces) who all received PAS and streptomycin in combination. In this paper each pneumothorax-treated lung is reported as a separate case.

The number of patients for whom artificial pneumothorax was considered indicated was, at the beginning of the study, about 25 per cent of the total number admitted to the sanatorium. During the latter half of 1949 and in 1950 this figure fell to a low record of 10 per cent, but increased thereafter to 17-18 per cent (Table II). Even when the pneumothorax inductions are calculated as a percentage of the patients hospitalized for the first time with tuberculosis—which possibly is more accurate—the figure shows a pronounced diminution since 1948-49. This decrease was at first chiefly due to the temporary narrowing of the indications for pneumothorax in

TABLE I

Age and Sex Distribution of Pneumothorax Cases

Group of Treatment	Number of Pneumothorax-treated Lungs	
	M.	F.
No Chemotherapy	48	68
PAS	39	43
PAS + Streptomycin	52	33

Table I (Continued)

Age and Sex Distribution of Pneumothorax Cases

Group of Treatment	AGE IN YEARS								Number of Patients	
	<16		16-25		26-35		36-55			
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
No Chemotherapy	—	1	18	23	16	26	12	9	46	59
PAS	—	—	13	18	13	20	13	5	39	43
PAS + Streptomycin	2	—	20	11	12	15	12	7	46	33

the hope that antimicrobial agents in combination with routine sanatorium care would suffice for cure in many mild cases. Since this hope was largely disappointing the lower figures in the more recent period were due to wider indications for thoracoplasty at the expense of pneumothorax.

Indications for Artificial Pneumothorax:

The main indication for this type of collapse therapy was fresh infiltration with caseation, usually in the upper lobe, but not infrequently in the apical segment of the lower lobe. Cavitation was considered definitely to be present when the roentgenograms showed well defined areas of thinning, 1 cm. or more in diameter. Suspected cavitation was not always investigated tomographically, particularly during the first period of the study, since at that time infiltration alone, especially with a tendency to recurrence, and the presence of tubercle bacilli on direct sputum smears were considered adequate indications for pneumothorax. Hence the higher frequency of cavity in the PAS and PAS-streptomycin groups (Table III) is ascribable both to improved diagnostic technique and somewhat stricter indications for pneumothorax. The possibility that a more favorable course in these latter groups might be due to selection of milder cases is precluded by the greatest percentage with cavity.

Before the introduction of antimicrobial agents pneumothorax induction generally was performed soon after the patient's admission to the hospital. But when it became apparent that PAS could control the disease[4] and so probably reduce the risk of pleural effusion the approximate length of the hospital stay prior to induction increased from three weeks to two months (Table IV) during which time specific medication was systematically administered.

TABLE II

Total number of patients admitted, number hospitalized for tuberculosis for the first time, and number of attempts at pneumothorax induction during the period January 1, 1948 to December 31, 1951. (The figures in brackets give the numbers in percentage of the total admissions)

Period	Total Admissions	First Admissions		Attempts at Pneumothorax	
		No.	Pct.	No.	Pct.
1/1-30/6, 1948	226	82	(36)	64	(28)
1/7-31/12, 1948	209	39	(19)	48	(23)
1/1-30/6, 1949	264	72	(27)	74	(28)
1/7-31/12, 1949	262	59	(23)	41	(16)
1/1-30/6, 1950	280	60	(21)	38	(14)
1/7-31/12, 1950	277	68	(25)	27	(10)
1/1-30/6, 1951	284	75	(26)	51	(18)
1/7-31/12, 1951	261	72	(28)	46	(18)
TOTAL	2,063	527	(26)	389	(19)

Table IV also shows that the total average hospital stay, instead of being prolonged by the delay in induction, decreased from 175 to 136 days. This may be attributed to a more rapid effect of the pneumothorax and above all to a lower complication rate.

Only in exceptional cases was pneumothorax in more recent times induced soon after admission. These were chiefly patients with cavity of the lower lobe, generally of its apex, and concurrent pleurisy which by pleural obliteration would shortly have ruled out pneumothorax, necessitating instead a major procedure such as lobectomy or segmental resection. In such cases pneumothorax was induced after one or two weeks of intensive specific medication. The results have been rapid disappearance of the effusion and a satisfactory selective collapse, usually after freeing the apex of the lobe from adhesions.

Results

Preinduction Administration of Antimicrobial Agents:

Before the great advantages of its combination with streptomycin became known PAS was given alone in preparation for pneumothorax. The dosage was 10 Gm. of para-aminosalicylic acid daily, given orally as 70 per cent granulate. Combined medication was introduced during the latter

TABLE III

The Frequency of Cavity (1 cm. or more in diameter) at Pneumothorax Induction

Group of Treatment	Number of Pneumothoraces	Number of Lungs with Cavity	
		No.	Pct.
No Chemotherapy	116	64	(55)
PAS	82	53	(65)
PAS and Streptomycin	85	66	(78)
TOTAL	283	183	(65)

TABLE IV

Average Duration of Hospitalization

	No Chemotherapy		— — PAS — —		Pas Plus Streptomycin	
	Number of Cases	Days in Hospital	Number of Cases	Days in Hospital	Number of Cases	Days in Hospital
Before Induction*	105	20	79	54	83	56
After Induction**	67	153	60	117	61	90
Average Total Period**	67	173	60	168	61	138

*These figures do not include the waiting period before induction of the second pneumothorax in patients with bilateral pneumothorax.

**These figures do not include:
a) Patients whose pneumothorax was abandoned for conservative or surgical therapy.
b) Patients in whom contralateral pneumothorax was induced.
c) Patients who received surgical treatment of the contralateral lung.

part of 1950 and has since been given as routine, with PAS in the above dosage and 1 Gm. of dihydrostreptomycin three times weekly. With few exceptions medication was given for about six weeks prior to pneumothorax induction and was continued for some time afterwards, at least until after adhesion section. The mean postinduction duration of medication in the PAS group was 106 days. In the PAS-streptomycin group PAS was given for a mean period of 74 days and the average quantity of streptomycin in that time was 20 Gm.

Following premedication the number of sputum conversions prior to induction was increased and the amount of sputum diminished. Figure 1 shows that 47 per cent of the untreated patients were fairly constantly sputum-positive at induction (i.e. showed tubercle bacilli in three or more of five consecutive direct smears). The corresponding figure in the PAS group was 22 per cent and in the PAS-streptomycin group only 13 per cent.

The influence of premedication on the erythrocyte sedimentation rate is also seen in Figure 1. While a sedimentation rate (Westergren) of more than 20 mm. in one hour was recorded immediately before pneumothorax induction in 59 per cent of the untreated group, the respective figures for the other groups were 22 per cent and 16 per cent.

The roentgenograms showed evidence of regression of the lesions prior to induction in the majority of the treated cases—66 per cent of the PAS group and 79 per cent of the PAS-streptomycin group (Figure 2). Progres-

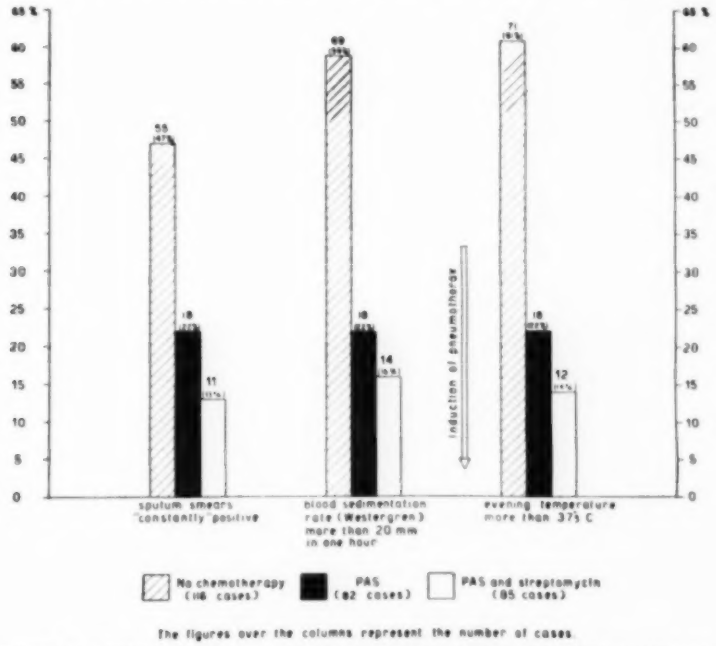


FIGURE 1: Sputum smears and blood sedimentation rate (Westergren) at the time of pneumothorax induction, and temperature after induction.

sion was observed in 16 and 3 per cent respectively and the disease in the remaining cases was stationary.

The shorter mean period of preinduction hospitalization in the untreated patients implies that only a limited number had serial roentgenograms taken in this period. And this number was mainly composed of the more severely ill patients. With this reservation the roentgenographic findings in the untreated patients who were hospitalized for more than one month before pneumothorax induction are presented in Figure 2 along with the data from the treated patients.

The Course of Pneumothorax Therapy After Premedication:

It appears logical to expect that a period of specific medication prior to pneumothorax induction should have a favorable influence on several complications which previously had to be reckoned with. If the progress of the disease is arrested, the exudative component controlled, and the patient thereby in better general condition with more normal sedimentation rate and fewer tubercle bacilli, the exacerbation of the disease which is the cause of the complications should be less likely to occur. The results of the

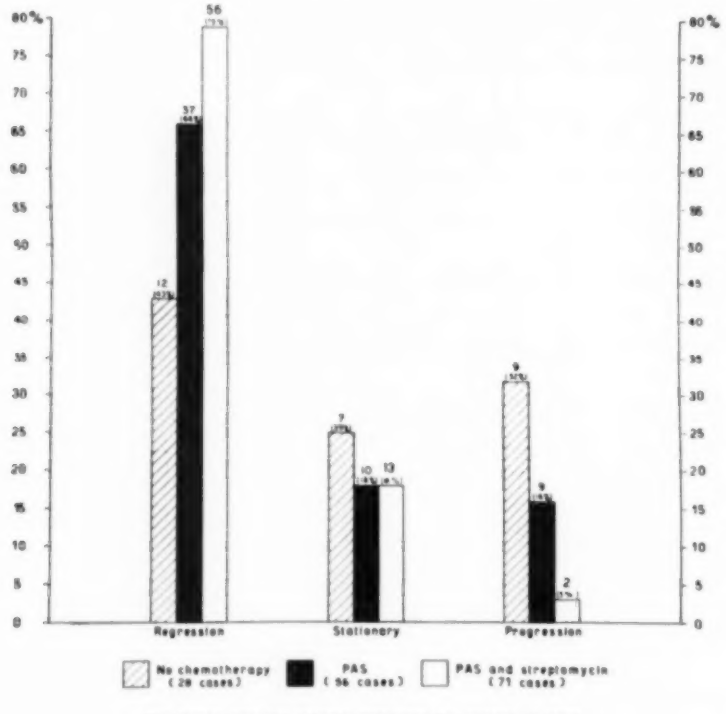


FIGURE 2: The lung roentgenograms prior to pneumothorax in patients hospitalized for more than one month before induction.

administration of antimicrobial agents in preparation for surgical collapse measures[1,2] provide unqualified support for this assumption.

Tension cavities following the establishment of artificial pneumothorax are now practically never seen. This is obviously because tuberculous lesions in the draining bronchus are brought by specific medication to a healing stage and the consequent reduction of swelling lessens the risk of a valve mechanism. Acute spread of the disease, which formerly occurred from time to time in association with pneumothorax induction, was never met with in the treated patients.

In earlier years the induction of artificial pneumothorax was commonly followed by pyrexia, which in all probability was caused by release of toxin from the collapsed lung. In this study postinduction evening temperature of more than 37.5 degrees C. was noted in 61 per cent of the patients who had not received antimicrobial agents. This figure fell to 22 per cent in the PAS group and 14 per cent in the PAS-streptomycin group (Figure 1). The lessened toxicity of the disease was, of course, the explanation of this difference.

The Formation of Pleural Adhesions:

One of the reasons for which we formerly refrained from allowing a period of conservative therapy prior to commencing artificial pneumothorax was the fear that pleural adhesions might form in this interval and prevent effective collapse. Later it was thought that this risk would be considerably lessened by the administration of antimicrobial agents. Such treatment, by preventing the spread of the disease to the pleurae, could be expected to decrease the likelihood of inflammatory pleural reactions, both productive in the form of adhesions and exudative in the form of effusion. In order to judge if the incidence of adhesions had been affected by systematic premedication the occurrence and extent of adhesions were noted in all cases in which pneumothorax was attempted (Table V).

The proportion of cases in which a free space was found was equal in the untreated and the PAS-streptomycin groups, viz. about 12 to 14 per cent. In the patients who received PAS alone the figure was only about

TABLE V

The Frequency of Pleural Adhesions and Their Significance for Pneumothorax Therapy in the Different Groups

Group of Treatment	NUMBER OF PNEUMOTHORAX LUNGS					
	No Chemotherapy		PAS		PAS and Streptomycin	
No adhesions	14	(12)	7	(8)	12	(14)
Lung freed by adhesion section	35	(30)	40	(49)	36	(42)
Adhesion section incomplete, pneumothorax continued	33	(29)	18	(22)	19	(22)
Pneumothorax abandoned within six months	34	(29)	17	(21)	18	(21)
TOTAL	116		82		85	

8 per cent. The difference—if it is significant—may conceivably imply that adhesions formed more frequently during PAS premedication but were prevented from doing so by the combination PAS-streptomycin. But as in most of the PAS cases the adhesions could be severed, the proportion of free lungs in this group finally exceeded that in the untreated group.

The lung was free either primarily or after adhesion section in 42 per cent of the untreated group, in 57 per cent of the PAS group, and 56 per cent of the patients who had received both PAS and streptomycin. In approximately 25 per cent of the remaining patients in all three groups the adherent lung could be freed to such an extent that a satisfactory pneumothorax could be maintained. The pneumothorax was abandoned within six months of induction in 29 per cent of the untreated patients and 21 per cent of those who had received antimicrobial agents.

Pleural Effusion After Pneumothorax Induction:

The definition of pleural effusion varies widely. In the present paper the term is applied to all collections of fluid in the pleural cavity—irrespective of other signs of inflammation—in excess of the minimal amounts which often appear periodically during the course of an artificial pneumothorax and appear to have no great significance. Thus effusions which filled the costophrenic sulcus but did not reach the dome of the diaphragm were not recorded. In the interests of uniformity, however, all the effusions noted in this study were observed until their complete disappearance.

"Spontaneous" effusion was the term applied to fluid unassociated with adhesion section, i.e. which appeared prior thereto or more than two months afterwards. Effusions which developed within these two months were considered to be caused by the intervention.

The frequency of spontaneous effusion in the patients who had received no antimicrobial medication was 15 per cent. After the introduction of PAS this figure was reduced to 9 per cent. Of the patients given combined PAS and streptomycin only 5 per cent developed spontaneous effusion

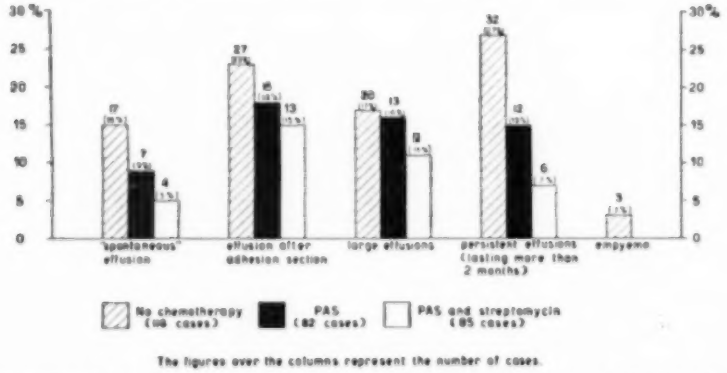


FIGURE 3: Incidence of serous pleural effusion and empyema during the first six months of pneumothorax therapy.

(Figure 3). Hence there can be no doubt that antimicrobial agents decreased the risk of this type of effusion.

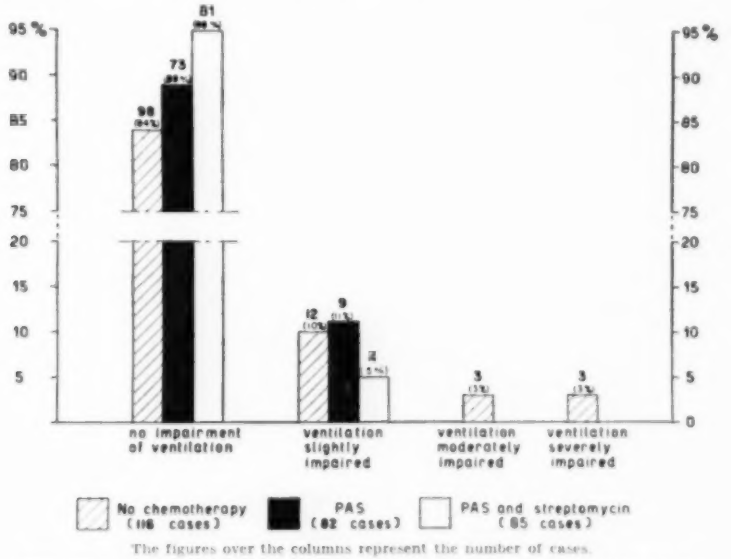
Pleural Effusion After Adhesion Section:

One of the highest hopes attached to specific medication in association with pneumothorax therapy was that it would counter the risk of dangerous pleural reactions to adhesion section, e.g. empyema or long-standing effusion with pleural thickening and reduced diaphragmatic mobility giving rise to ventilatory insufficiency.

The number of adhesion sections in this series gradually increased as it was shown that antimicrobial agents justified this hope. Latterly more extensive freeing has been undertaken and blunt dissection in the extrapleural connective tissue increasingly employed. With each intervention of multiple stage sections counted separately the frequency of this operation was as follows: 90 adhesion sections on 116 lungs (78 per cent) in the untreated group, 68 on 82 lungs (83 per cent) in the PAS group, and 77 on 85 lungs (91 per cent) in the PAS-streptomycin group.

The incidence of pleural effusion following adhesion section in these three groups showed a difference which, although not great, was quite clear, viz. 23, 18 and 15 per cent respectively (Figure 3).

It was found that the effusions which formed in the patients who had received antimicrobial agents were more benign in behaviour. Large collections of fluid (both spontaneous and associated with adhesion section) were more common in the untreated group—17 per cent, than in the PAS-streptomycin group—11 per cent. Combined medication, therefore,



The figures over the columns represent the number of cases.

FIGURE 4: Roentgenological assessment of ipsilateral ventilation six months after pneumothorax induction (all patients).

appeared to have accomplished a certain reduction in the severe pleural reactions which produce copious effusion. PAS alone was not found to have that effect in this study.

The most interesting point in this connection, however, is that the effusions which developed in the patients who had received specific medication reabsorbed more rapidly than those in the untreated patients. In this series an effusion which had not entirely disappeared after two months was termed persistent. The difference in the number of persistent effusions was the most striking in the comparison of the three groups (Figure 3). From being 27 per cent in the untreated patients the frequency of this complication, so disastrous from the aspect of respiratory function, fell to 7 per cent in the PAS-streptomycin group.

Empyema:

Since the introduction of our present methods pleural empyema has never been met with in the first six months of pneumothorax therapy. Three cases (1 mixed infection) were recorded in 1948 in patients who had not received antimicrobial agents. The frequency of empyema, therefore, was 3 per cent in the untreated cases and none in the treated. In none of the three patients with empyema did the complication appear to be attributable to adhesion section.

Impairment of Function Due to Artificial Pneumothorax:

There being no exact method for determining the function of a lung collapsed by artificial pneumothorax an attempt was made to assess the results in this respect six months after induction with the aid of the roentgenogram. Particular consideration was paid to two factors. One of these, fibrin deposition of the surface of the lung with thickening of the pleura hindering free expansion was of lesser significance in the short observation period of this study. The other factor was the mobility of the diaphragm.

Cases of impaired ventilatory function were classified into three grades:

1) *Slightly impaired ventilation* with minor adhesions between the diaphragm and chest wall in the costophrenic sulcus or an insignificantly thickened pleura over a lung capable of good expansion.

2) *Moderately impaired ventilation* with complete obliteration of the costophrenic sulcus and more reduced, but still fairly good, diaphragmatic mobility.

3) *Severely impaired ventilation*, finally, denoted severe reduction of diaphragmatic mobility. In these cases there generally was also some pleural thickening.

It was not possible, of course, to draw distinct dividing lines between these three degrees. But the assessment was made without knowledge of the group to which the patient belonged.

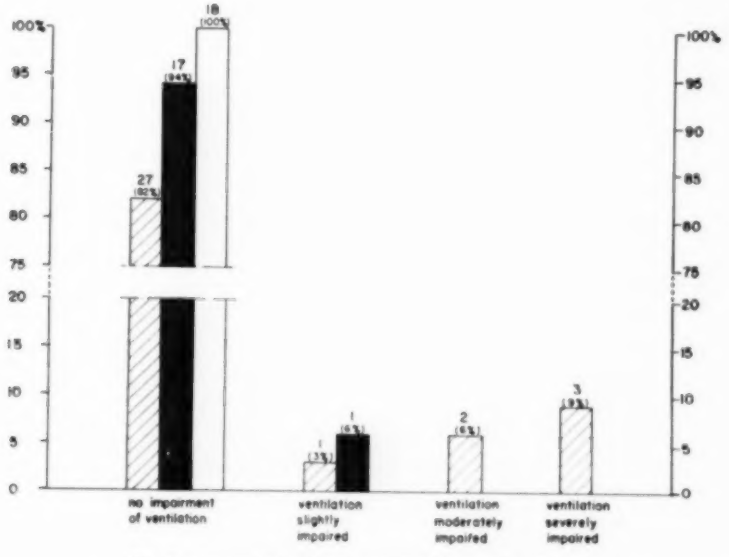
A survey of all the patients in the study, including those in whom the pneumothorax was abandoned during the observation period, shows that better functional results were obtained in those given specific medication

(Figure 4). Completely satisfactory function was judged to be present in 84 per cent of the untreated group while the figures for the treated groups were 89 and 95 per cent. Further, it is significant that moderate and severe degrees of functional impairment were confined to the untreated patients.

Study of the patients whose pneumothorax was abandoned within six months of induction shows that six of the 33 who had not received specific medication had more or less severe impairment of function; only one of the 18 PAS cases had slight impairment and no damage to function was observed in any of the 18 PAS-streptomycin cases (Figure 5).

Discussion

Since artificial pneumothorax technique in the period covered by this study underwent a double modification by means of antimicrobial agents and the "guided symphysis" treatment of effusion, one may wonder to what degree these factors were individually responsible for the improvements herein reported. In regard to spontaneous effusion, the two-thirds reduction was, of course, attributable only to antimicrobial agents. The more rapid disappearance of pleural fluid, however, may also have been influenced by the guided symphysis therapy. It is known that frequent aspiration, which forms part of this technique, by itself can shorten the duration of an effusion.[5] Guided symphysis, therefore, must certainly have contributed to the retention of ventilatory function in cases in which pleural thickening would otherwise have occurred and left an unexpandable lung.



The figures over the columns represent the number of cases.

FIGURE 5: Ipsilateral ventilation six months after induction in patients whose pneumothorax was abandoned during that period.

The results of this study indicate that, with the aid of the new specific drugs, artificial pneumothorax can be undertaken without the great risks of pleural effusion and empyema which have given this form of collapse a poor reputation in so many quarters. The frequency of empyema during the first six months of pneumothorax was shown to be reducible to none. And it is scarcely likely that in any but very exceptional cases effusions forming later in the course of therapy—provided they are properly treated—should progress to empyema. Although resection procedures are being increasingly performed in patients who would otherwise have been candidates for artificial pneumothorax, the restricted availability of surgical measures ensures that a considerable time must elapse before they can be generally employed. If artificial pneumothorax can be undertaken without the earlier fears of empyema and permanently impaired ventilation following pleural effusion, it will well be able to defend its place in the therapeutic arsenal against tuberculosis.

SUMMARY

An investigation is presented of the effect of antimicrobial agents on artificial pneumothorax therapy. Three groups of patients were observed for six months after the induction of pneumothorax:

A) One hundred sixteen consecutive pneumothoraces without specific medication—the control cases.

B) Eighty-two consecutive pneumothoraces in which all but seven of the patients had received para-aminosalicylic acid both before and after induction.

C) Eighty-five consecutive pneumothoraces with PAS and dihydrostreptomycin given in combination both before and after induction.

1) A free space was found at induction or was obtained after adhesion section in 42 per cent of group A, 57 per cent of group B, and 56 per cent of group C.
2) "Spontaneous" pleural effusions formed in 15 per cent of group A, 9 per cent of group B, and 5 per cent of group C.
3) Adhesion section was followed by pleural effusion in 23, 18, and 15 per cent of the respective groups.
4) Pleural effusion persisted for more than two months in 27, 15 and 7 per cent.
5) Empyema developed in 3 per cent of the patients in group A but in none in groups B and C.
6) Impairment of ventilatory function due to pleural effusion was observed in 16 per cent of group A; in a number of these cases the impairment was severe. In groups B and C only 11 and 5 per cent showed any functional damage and this was invariably slight.
7) In addition to antimicrobial agents the patients in groups B and C who developed pleural effusion were treated with the writer's technique of "guided symphysis" and this contributed to the lack of functional impairment.

RESUMEN

Se presenta el resultado de una investigación sobre el efecto de los agentes antimicrobianos en el neumotorax terapéutico. Se observaron tres grupos de enfermos por seis meses después de iniciado el neumotórax:

A) Ciento dieciseis casos de neumotorax sin medicación especifica como grupo de control.

B) Ochenta y dos casos consecutivos de neumotorax en los que, con excepción de 7, todos los demás recibieron ácido paraminosalicílico antes y después de iniciado el neumo.

C) Ochenta y cinco casos de neumotórax con PAS y dihidroestreptomicina en combinación, antes y después de iniciado el neumo.

1) Se encontró espacio libre o se obtuvo después de seccionar adherencias en 42 por ciento del grupo A, 57 por ciento del grupo B y en 56 por ciento del grupo C.
2) Derrames pleurales "espontáneos" se formaron en 15 por ciento del grupo A, 9 por ciento del grupo B y 5 por ciento del grupo C.
3) La sección de adherencias fué seguida de derrame pleural en 23, 18 y 15 por ciento de los grupos respectivos.
4) El derrame pleural persistió por mas de dos meses en 27, 15 y 7 por ciento de los grupos en el orden mencionado.
5) El empiema se desarrolló en 3 por ciento de los enfermos del grupo A pero en ninguno de los grupos B y C.
6) Reducción de la función ventilatoria debida a derrame pleural se observó en 16 por ciento del grupo A; en cierto número de estos casos la reducción fué severa. En los grupos B y C solo 11 y 5 por ciento mostraron algún daño pero este fué invariablemente ligero.
7) Ademas del uso de los agentes microbianos en los grupos B y C que desarrollaron derrame pleural el autor los trató con su técnica de "sínfisis conducida" y esto contribuyó a la falta de daño funcional.

RESUME

L'auteur rapporte ses recherches sur l'effet des agents antimicrobiens sur le traitement par le pneumothorax artificiel. Trois groupes de malades furent observés pendans six mois après la création du pneumothorax:

A) Le grupe témoin est constitué par 116 pneumothorax auxquels n'a été adjointe aucune médication spécifique.

B) 82 pneumothorax reçurent de l'acide para-aminosalicylique avant et après la création du pneumothorax sauf pour 7 d'entre eux.

C) 85 pneumothorax eurent en même temps du P.A.S. et de la dihydrostreptomycine associée, avant et après leur création.

1) On obtint un décollement complet, soit à la création, soit après section de brides dans 42% des malades du groupe A, 57% du groupe B et 56% du groupe C.
2) Des épanchements pleuraux spontanés se constituèrent dans 15% des malades du groupe A, 9% du groupe B et 5% du groupe C.

3) La section de brides fut suivie d'un épanchement pleural dans 23, 18 et 15% des groupes respectifs.
4) Les épanchements pleuraux persistèrent plus de deux mois dans 27, 15 et 7% des groupes.
5) Une pleurésie purulente survint chez 3% des malades du groupe A mais dans aucun des groupes B et C.
6) Des troubles de la fonction respiratoire consécutifs à l'épanchement pleural survinrent chez 16% des malades du groupe A et parmi ceux-ci, certains furent très importants. Dans les groupes B et C, il n'y eut que respectivement 11 et 5% de troubles fonctionnels, et ceux-ci furent toujours légers.
7) Associé aux médications antimicrobiennes, les malades des groupes B et C atteints d'épanchement pleural subirent le traitement décrit par l'auteur sous le nom de "symphyse guidée," ce qui contribua au maintien d'un bon état fonctionnel.

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Congenital Anterior Chest Wall Deformities of Diaphragmatic Origin

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Deformities of the anterior chest wall are congenital or acquired. The acquired group of deformities result either from trauma or disease. The congenital group consist mainly of those deformities that result from failure of sternal fusion or malformation of the sternum, ribs or vertebrae. It is the author's opinion[2-4] that the three deformities of the anterior chest wall commonly known as funnel chest, pigeon chest and Harrison's grooves are congenital and are produced by the abnormal inspiratory contractions of an abnormally developed diaphragm. Through the years each of these three deformities has been described under a large variety of names for which the author has suggested the following:

1) Congenital chondrosternal depression for the "funnel chest," etc.
2) Congenital chondrosternal prominence for the "pigeon chest," etc.
3) Congenital chondrocostal grooves for Harrison's grooves.

Similarly many causes for their production have been described. The most common of these have been rickets, obstructive respiratory conditions, prolonged chronic dyspnea and cough, and abnormal intra-uterine pressure against the anterior chest wall. In the literature each author has presented his favorite name for the deformity, arguments for his etiologic theory and a case or two to support it. This article will describe the development of these deformities and present a theory to explain their production.

In previous publications,[2-4] existing beliefs concerning the etiology of these deformities have been challenged and the diaphragmatic theory as the cause for all three of these related conditions has been suggested. For years rickets was the most popular accepted cause for all three deformities until it was shown by careful biochemical studies that, in the majority of these patients, rickets did not exist and the deformities were not affected by intensive anti-rachitic treatment. The theory that obstructive respiratory lesions were responsible for them has been supported largely by the fact that, in infants, each is aggravated during forceful inspiratory efforts such as occur during crying, coughing or attacks of dyspnea. However, it is common to see normally developed chests in the presence of such obstructive lesions and to see infants with these deformities unaccompanied by similar obstructive lesions. The same facts hold true for the theory that chronic prolonged dyspneic states or cough are responsible. To negate this belief it needs only to be pointed out that the majority of children with congenital cardiac defects or chronic pulmonary disease have normally developed chests. Conversely, these deformities often exist in children with normal hearts and lungs. The intra-uterine compressive

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theory has never been proven but will be satisfactorily refuted in this discussion.

The theory that a congenital altered diaphragm is responsible is supported by certain observations and facts that are more convincing than the theories now in existence. These will be discussed first generally and then specifically as they apply to each deformity. It must be emphasized at this point that in the infant these deformities are mobile and are created or increased only during inspiration whereas in children and adults they are practically immobile and have a definite anatomic pattern. In a certain number of infants and children they are present to such mild degree that one must consider them within the normal range beyond which these deformities exist in varying degrees from mild to severe. However, at this point it is advisable to review certain anatomic and physiologic facts about the diaphragm as they affect and produce not only the normal contour of the chest but also these deformities.

Development of the Diaphragm

The normal diaphragm consists of a circumferential musculature with radiating muscle bundles taking their origin at the costochondral attachments and a central tendinous trefoil membrane into which the muscle fibers insert. If we compare the developed diaphragm (Figure 5) with the diaphragm (Figure 4) of a 9 mm. embryo[1] the following facts may be noted. The anterior portion of the diaphragm, which is derived from the septum

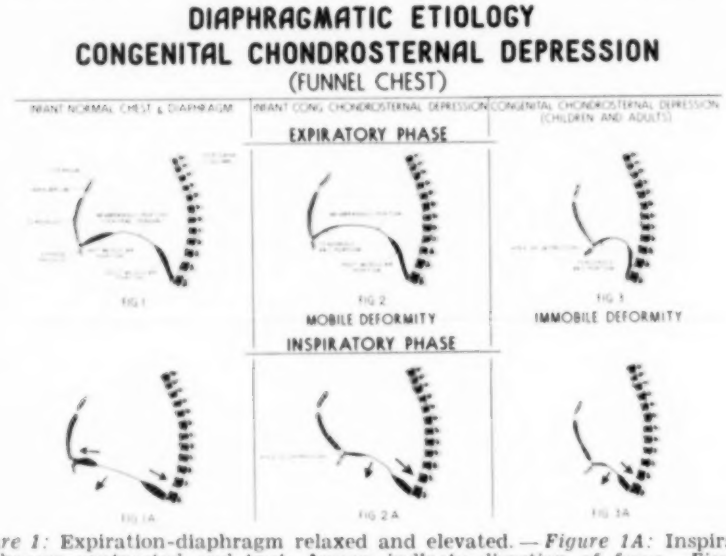

Figure 1: Expiration-diaphragm relaxed and elevated. — *Figure 1A:* Inspiration-diaphragm contracted and taut. Arrows indicate direction of force.—*Figure 2:* Expiration-diaphragm relaxed. Chest normal in appearance.—*Figure 2A:* Inspiration-diaphragm contracted. Anterior chest wall retracted by the pull of the contracting diaphragm. Note arrows.—*Figure 3:* Expiration-diaphragm relaxed. The anterior retracted chest wall is fixed. — *Figure 3A:* Inspiration-diaphragm contracted. The deformed anterior chest wall is fixed.

transversum of the embryo, arises on the posterior surface of the gladiolar-xiphoid junction and the cartilages of the sixth ribs. Its muscle fibers are inserted into the anterior leaf of the trefoil tendinous membrane. Separated from the anterior portion by a raphe are the two lateral portions of the diaphragm derived from the pleuro-peritoneal membranes of the embryo. The muscle fibers of the lateral portions arise at the costal attachments of the seventh to the eleventh ribs and are inserted into the outer margins of the two lateral leaflets of the trefoil membrane. Separated from the lateral portions by a fibrous triangular raphe, known as the costovertebral trigone, is the posterior portion, which is derived from the dorsal mesentery of the embryo. This posterior portion arises at the lower medial border of the twelfth rib and, after being joined by the two crurae, is inserted into the posterior and medial margins of the lateral leaves of the trefoil membrane.

Anatomic and Physiologic Considerations

In infants, both domes of the diaphragm are comparatively flat and occupy a relatively high position in the thorax. The costophrenic sinuses are shallow posteriorly and laterally and absent anteriorly.[5] The anterior muscular portion of the diaphragm, which is smaller, less muscular than the other portions, is practically perpendicular to the anterior chest wall. Any pull on the part of the contracting diaphragm would therefore be more quickly and strongly felt at its anterior attachment. In spite of these facts, contractions of the normal diaphragm will not retract the anterior chest wall. However, if this anterior portion is deficient in muscular fibers or underdeveloped, the contracture of the remaining portion of the diaphragm will affect a pull on this anterior portion as on the rest of the membranous portion of the diaphragm and will retract the anterior chest wall during inspiration. This fact must be constantly kept in mind in order to comprehend the inspiratory retraction of the anterior chest wall in infants

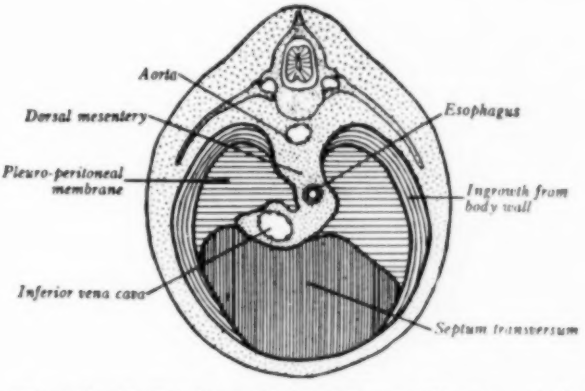

FIGURE 4 (From Arey's Developmental Anatomy): Diagram of diaphragm of 9 mm. embryo. (1) Septum Transversum which becomes the anterior segments. (2) Pleuro-peritoneal membranes which form the lateral segments. (3) Dorsal mesentery which forms the posterior segment. (4) Muscular ingrowth from body wall which contributes to the circumferential muscular portion.

with these deformities and its evolution into the fixed type of deformity in later life. The particular variation of this anterior portion of the diaphragm is the determining factor as to which of these deformities will be produced, as well as its extent.

The contraction of a normally developed diaphragm acts like any other muscle. For example, when the biceps contracts, its point of origin is stationary and its point of insertion is moved producing a flexion of the forearm. Similarly, when the diaphragm contracts, its origin along the periphery of the thoracic outlet is stationary, whereas its insertion, the central trefoil membranous portion, is pulled down. The diaphragm as a whole becomes taut and descends to its inspiratory position without any pull on any portion of its origin along the periphery of the thoracic cage (Figures 1 and 1A). Thus a normally developed infant shows no retraction during quiet respiration and the diaphragm descends inflating the lungs and pushing the abdominal contents downward and forward. However, during unusual inspiratory efforts as crying spells or hiccoughing typical Harrison's grooves are visible as a result of the inspiratory retraction of the anterior sixth ribs, cartilages and adjacent interspace due to the stronger spastic contractions of the diaphragm. Under similar circumstances, inspiratory retraction of the chondrosternal area may be seen as a result of unusually strong contractions of the diaphragm and the relative weakness of the

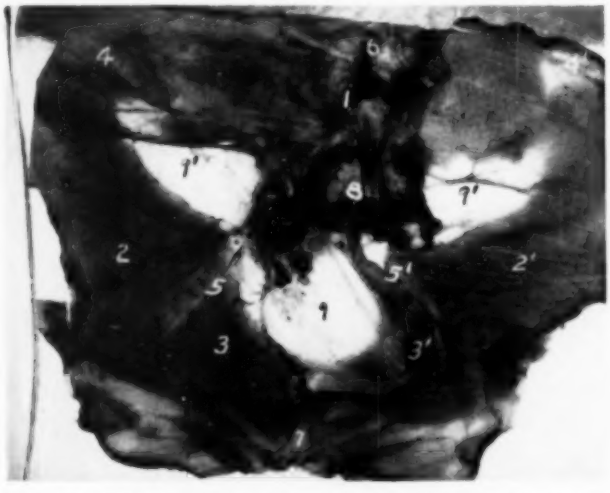

FIGURE 5: Normal diaphragm from a normal infant for correlation with Figure 4. (1) Posterior segment. (2, 2') Two lateral segments. (3, 3') Anterior segments derived from the septum transversum. (4, 4') The costovertebral trigones separating the lateral segments from the posterior segment. (5, 5') The raphe separating the lateral segments from the anterior segments. (6) Vertebral column. (7) Gladiolar-xiphoid junction of the sternum. (8) Esophagus. (9, 9') Anterior and lateral leaves of the trefoil membranous portion of the diaphragm. The costochondral circumference is the origin of the diaphragmatic muscle fibers and the trefoil membranous portion is the tendinous insertion of these muscle fibers. Contraction of the diaphragmatic circumferential muscle creates a force of pull down and toward the costochondral circumference.

anterior portion of the diaphragm compared to the remaining diaphragmatic musculature. For example, a 26 week, four pound premature infant with a congenital tracheo-esophageal fistula had typical chondrosternal inspiratory retractions whenever the excessive mucus obstructed the larynx. This disappeared when the mucus was removed by suction and the chest appeared normal. This occurred because the diaphragmatic contractions were unusually strong and the weaker and smaller anterior portions of the diaphragm yielded to the stronger lateral and posterior portions. The autopsy showed a normally developed chest and diaphragm.

At birth, the transverse and antero-posterior diameters of the chest are practically equal. During infancy, the contraction of the diaphragm is chiefly responsible for converting the infantile round chest into the normal elliptical shaped chest of the child. The ribs in the infant are practically horizontal. As the infant develops, definite changes occur in the contour of the chest. The ribs grow out laterally and downward and there is a progressive elongation and flattening of the thorax during the first five years of life. The costophrenic sinuses become deeper as the diaphragmatic attachments follow the downward growth of the ribs. The auxiliary muscles of respiration of the thorax develop and help in the expansion of the thorax during inspiration. Thus the normally developed diaphragm in the normal infant produces no retraction of the anterior chest wall but assists in the further development of the normal elliptical contour of the mature chest.

Common Characteristics

The three deformities under discussion have certain common characteristics:

1) In infants each is characterized by an inspiratory retraction of some portion of the anterior chest wall.

2) Each is always present at birth and appears with the first inspiration. If not observed at birth it has been overlooked, giving rise to the erroneous impression that it developed months or years later.

3) The inspiratory retraction of the anterior chest wall is synchronous with the inspiratory contraction of the diaphragm.

4) These deformities are usually bilateral but may be unilateral.

5) They vary in extent from mild to severe.

6) Each deformity is mobile during the first few years of life when the anterior chest wall is soft and yielding.

7) In infants, the retractions are aggravated by stronger inspiratory efforts as in crying or attacks of dyspnea or cough.

8) If the hemi-diaphragm of an infant with any one of these deformities is paralyzed by the injection of a few cc. of 1 per cent novocaine into the phrenic nerve in the neck, the inspiratory retraction on that side will cease for the duration of the anesthetic effect.

9) As the chest wall becomes stronger and more rigid, each deformity becomes less mobile and assumes its characteristic fixed pattern in the position of inspiratory retraction.

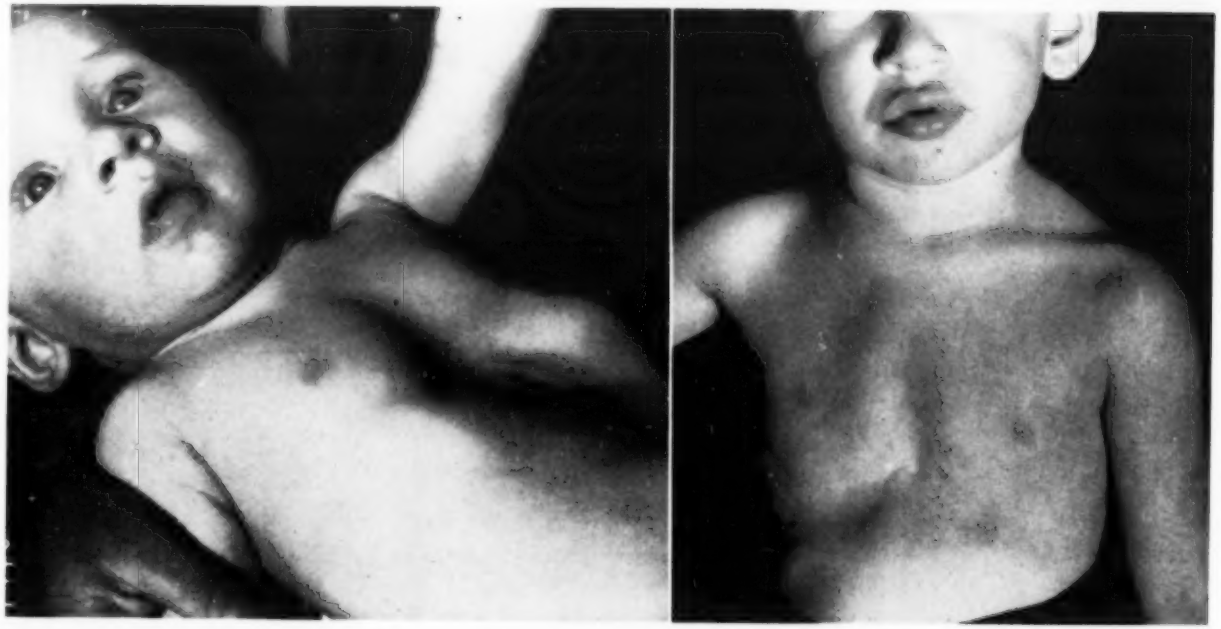

FIGURE 6 FIGURE 6A

Figure 6: Congenital Chondrosternal Depression (funnel chest). Male infant, age five months, normal and healthy in all respects except for severe inspiratory retraction of the anterior chest wall and upper abdomen first noted at birth. Diagnosed and treated for rickets without effect. Phrenosternolysis performed at age of five months with immediate improvement.—*Figure 6A:* Same infant two years later. Note small curved transverse incision and normal well developed chest.

Congenital Chondrosternal Depression (Funnel Chest)

This deformity, which consists of a depression of the chondrosternal area with its deepest point at the gladiolar-xiphoid junction has been described in the literature since the sixteenth century. It has been referred to by numerous names, the most common of which are funnel chest, pectus excavatum, trichterbrust, chone-chondrosternon, etc. The author[3,4] proposed the name congenital chondrosternal depression because it is more descriptive and relates to its congenital character. Ochsner and DeBakey[12] have reported an incidence of this deformity in 28 cases found in 46,703 persons examined or .06 per cent. The author[2] has reported a similar incidence of 25 cases in 41,963 Newark school children.

In order to understand this deformity better, one must appreciate the difference that exists between its mobile form in infants and its fixed form in children and adults (Figures 2, 3A). In infants, this deformity is usually first noted at birth. The depression is created during inspiration as though a hook were inserted into the dorsal surface of the gladiolar-xiphoid junction and pulled dorsally thus making the apex of the depression at this junction. The gladiolus and xiphoid portions of the sternum, adjoining cartilages and the epigastrium are drawn dorsally with it to produce the depression (Figures 2A and 6). During expiration the diaphragm relaxes, the pull is released and the depression is eliminated by the elastic recoil of the anterior chest wall (Figure 2). The depression is increased when inspiration is deeper as during crying spells or in the presence of hiccough.

As the infant grows older and the anterior chest wall becomes rigid, the retraction becomes more shallow as the depression becomes gradually fixed in the position of inspiratory retraction. During early childhood only forced inspiration will produce an increase in the depression. Usually after the age of three years it has become permanently fixed to a varying degree (Figures 3, 3A and 7). In children and adults the deformity consists of a fixed depression, the deepest portion of which is at the gladiolar-xiphoid junction. The gladiolus is gradually retracted posteriorly starting from the angle of Ludwig to the apex of the depression at its junction with the xiphoid. The sides of the depression consist of the angulated deformed cartilages usually from the third to the seventh.

For many years clinicians have held that this deformity was a manifestation of rickets but now some authors[7,11] concede that "some congenital anomaly" is the more likely cause. The aggravation of the depression during forced respiratory efforts and its temporary production during attacks of dyspnea in normal infants led others to believe that the deformity was caused by obstructive lesions in the upper respiratory tract. This theory is questioned not only because the depression is not affected after the correction of such obstructive respiratory conditions but also because the depression is more often present when no obstructive pathology exists. Recently some authors[5,8,11] have stated that the condition is a result of "a short central tendon" of the diaphragm. This is refuted because during expiration the chest recoils to a normal position and a short central tendon

FIGURE 7

FIGURE 7A

Figure 7: Male, age 17 years. Severe congenital chondrosternal depression with orthostatic pulse and blood pressure.—*Figure 7A:* Same patient one week after chondrosternoplasty with marked cosmetic improvement. Now serving in the army.

would not permit this. Furthermore fluoroscopic study of the contour and action of the diaphragm in the lateral position fails to show any such condition. It does reveal the inspiratory retraction of the anterior attachment of the diaphragm and gladiolar-xiphoid junction which can only be produced by the pull on the passive tendinous anterior portion. Unfortunately no one has described a diaphragm with this deformity. However, the tendinous underdeveloped anterior portion of the diaphragm of a four week old suckling puppy with a typical chondrosternal depression as is seen in humans, has been described.[3] During operations both in infants and the older group, it was observed that the anterior portion of the diaphragm as far as could be seen was largely fibrous in character. Therefore, the author is of the opinion that the cause for this deformity is not "a short central tendon of the diaphragm" but a deficiency of musculature or an arrest in the development of the anterior portion of the diaphragm which is derived from the septum transversum. When such a diaphragm contracts during inspiration, its anterior attachments no longer acts as a site of origin, but, because of its relative muscular weakness is pulled backward with the remaining membranous portion of the diaphragm, thereby retracting the attachment to the anterior chest wall. This is the mechanism for the mobile inspiratory retraction of the anterior chest wall in infants (Figures 2 and 2A). This anterior attachment of the diaphragm at the gladiolar-xiphoid junction becomes the apex of the fixed deformity in the child and adult (Figures 3 and 3A).

Infants with only congenital anterior chest wall deformities do not have cardiorespiratory disturbances in spite of the paradoxical motion of the anterior chest wall. When signs of cardiorespiratory embarrassment are present, other congenital anomalies, usually cardiac, are responsible. In children, with a fixed depression, because of greater physical activity and increased demands on the cardiorespiratory system, symptoms such as dyspnea on exertion, palpitation, precordial pain, easy fatigability and dizziness may appear. There is often no relationship between the depth of the depression and the presence or severity of cardiorespiratory symptoms. Although severe deformities are consistent with normal and strenuous activity, patients with even moderate depressions may show signs of interference with normal physical activity. How many of these reach old age is a question. Master and Stone[9] believe that these symptoms are "neurogenic or the result of inadequacy of the small heart in its response to emotional stress." However, when this type of patient has been treated by surgery and the depression corrected, not only have the symptoms disappeared but the patient was able to engage in strenuous activity without difficulty. These cardiac symptoms may be due to the effects of abnormal rotation of the heart and great vessels or the restriction and pressure on the heart by the depressed chondrosternal area. In the adolescent, particularly males, emotional symptoms and neuroses are often present because of the deformity. Mild cases may be relieved by reassurance and psychotherapy. Severe cases have been cured of their symptoms with surgical treatment.

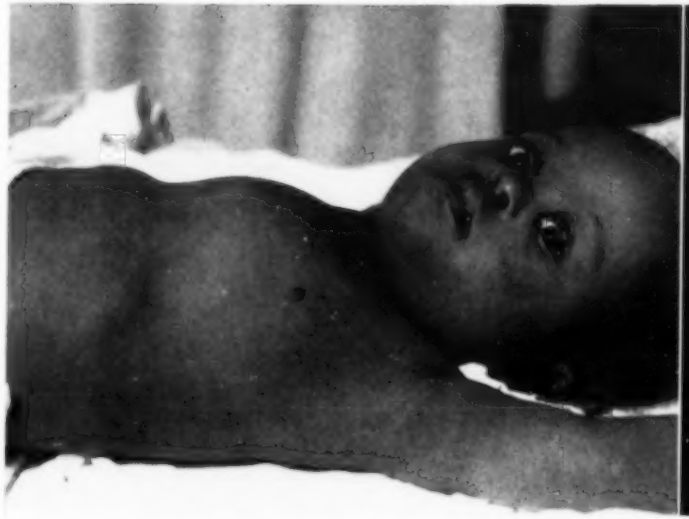

FIGURE 8

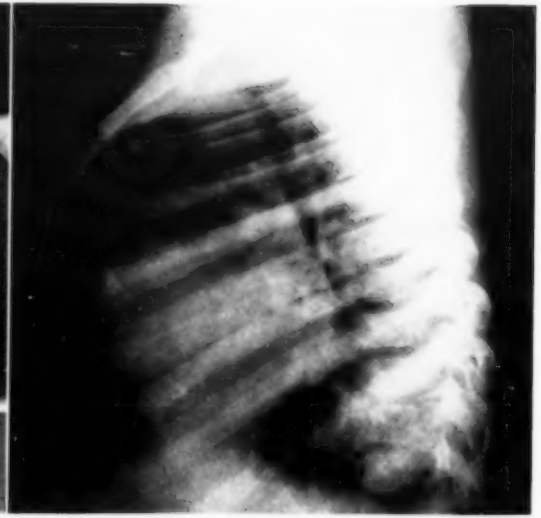

FIGURE 8A

Figure 8: Mongoloid, negro infant, age 14 months. Typical congenital chondrosternal prominence (pigeon breast). Upper thorax is round and lower thorax is elliptical and retracted. Note the horizontal grooves (Harrison's). — *Figure 8A:* Lateral x-ray view of same infant showing the persistence of the infantile rotundity of the upper thorax. The manubrium sterni maintains this position throughout life while the rest of the sternum retracts posteriorly to form the elliptical shape of the lower half of the thorax.

There are usually no physical signs in infants except the inspiratory retraction of the chondrosternal area of the chest. In children, one sees the thin asthenic type, the typical posture with the head held forward, shoulders rounded and the anterior chest wall flat and depressed. Tachycardia, sinus arrhythmia, apical systolic murmurs and often an orthostatic pulse and blood pressure are the most common cardiac signs. Of unusual interest has been the so-called orthostatic pulse and blood pressure seen quite frequently in the older group. In the standing position they had a rapid pulse and a comparatively low systolic blood pressure with a small pulse pressure. On lying down the pulse promptly diminished in frequency, the systolic blood pressure was elevated and the pulse pressure increased. The electrocardiographic abnormalities are low voltage, deviation of electrical axis, inverted T waves in leads three and four. Immediate postoperative electrocardiographic examination failed to show much change although symptoms and signs were greatly relieved.

Surgical Treatment

Surgical treatment is divided into two types, phrenosternolysis for infants with a mobile depression and a chondrosternoplasty for children and adults with a fixed depression. The only indication for the surgical treatment of the infant is to prevent or diminish the subsequent fixed depression of the chondrosternal area which would otherwise develop later. Theoretically, the earlier the infant is treated surgically the less deformity will result. However, the author has seen three new-born infants with distinct typical inspiratory retractions who after six weeks had normal immobile chests. One must conclude that in these three infants the anterior portion of the diaphragm was deficient at birth but continued to develop its muscle fibers sufficiently to function normally and thus eliminated the inspiratory anterior chest wall retraction. Although the youngest infant operated by the author was four months, it is now the practice to recommend surgery after three months of age provided the inspiratory retractions have shown no signs of diminution.

The procedure of phrenosternolysis consists in a modification of the procedure first developed by Brown[5] which consists in separating the diaphragmatic attachment from the sternum by first incising the gladiolar-xiphoid junction and resecting a portion of the xiphoid. In addition the fused sixth and seventh cartilages are resected a short distance from the sternum. The detached edge of the diaphragm is sutured to the freed edge of the rectus muscles. This not only diminishes the possibility of herniation but may provide muscle fibers to the anterior portion of the diaphragm for possible future development. This operation has been performed on 15 infants. The best results have been in the younger infants in whom the depression is most mobile. In the older infants, the less mobile the retraction the less satisfactory is the operation of phrenosternolysis. In such patients it is best to resect a portion of the deformed lower cartilages.

In children and adults with a fixed deformity, a chondrosternoplasty is necessary.[4] This procedure which was previously described has been modi-

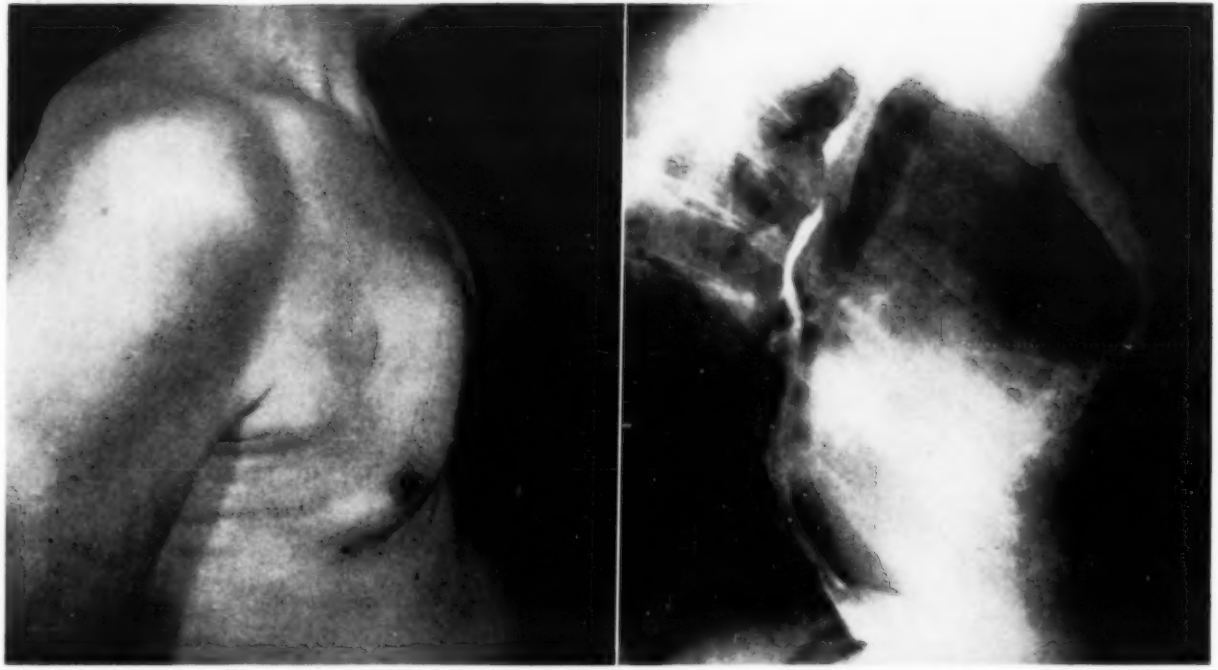

FIGURE 9 FIGURE 9A

Figure 9: The adult type of congenital chondrosternal prominence, present since birth. Note the relative prominence of the upper half of the thorax and the normal elliptical shape of the lower thorax. — *Figure 9A:* Lateral x-ray view of the same patient. Note the persistence of the infantile rotundity of the upper half. The prominence is the result of failure of the manubrium sterni to retract with the rest of the sternum.

fied. A vertical incision from the manubrium to the xiphoid is made and if a longer incision is necessary it is made along the costal arch. The pectoralis major muscles are separated from the third to the sixth cartilages. Small segments of these cartilages are excised at each side of the sternum. The sternum is cut horizontally just above the third cartilaginous junction. The xiphoid is separated from the gladiolus and the sternum is gradually elevated carefully avoiding opening the pleura. With the sternum elevated in the new position, more of the deformed cartilages are trimmed away to meet the medial ends of the cartilages. Instead of a free cartilage graft to support the elevated sternum, a narrow piece of tantalum mesh or wire is placed underneath the elevated sternum to serve as a support and sutured to the lateral edges of the sixth cartilages. The edges of the re-lined cartilages are sutured with steel wire sutures. Entrance into the pleural cavities can be avoided by careful stripping from the outer edge of the cut cartilage toward the mid line. Attempting to strip the pleura any other way invariably will open the pleural cavity. No external traction is used. To date fourteen chondrosternoplasties have been performed with very satisfactory results.

Congenital Chondrosternal Prominence

This deformity which occurs much less frequently has been described as a prominence of the sternum and the cartilaginous parts of the ribs with a depression of the sides of the thorax. It has been called pigeon chest, chicken breast, pectus carinatum, keel-shaped breast and pyriform chest. The name congenital chondrosternal prominence seems more descriptive for this deformity because it refers to its anatomic configuration and congenital origin. It is more readily co-ordinated with its opposite deformity, congenital chondrosternal depression, and it more adequately describes the deformity instead of comparing it to the shape of an object.

Just as for congenital chondrosternal depression, the same unsupported etiological factors have been offered to explain the production of congenital chondrosternal prominence. These likewise have been refuted in a previous publication.[3] Some authors[11] still consider this deformity a manifestation of rickets while others are beginning to question the rachitic etiology by stating "it is doubtful if this depends on rickets alone." Time will prove that rickets in most instances has been assumed rather than proven. In three infants and three adults with this deformity rickets was not a factor. It is of interest that among the three infants, two had congenital heart lesions and the third had severe dyspnea and cyanosis due to congenital agnathia. In the last, the deformity was not affected after the dyspnea and cyanosis were relieved by surgical treatment. One of the adults had a congenital absence of a hand. Observation and study of a fourteen month old infant[3] with this deformity furnished the first evidence that the deformity is congenital and that it is produced by the abnormal or uneven contractions of an abnormally developed diaphragm. This infant (Figure 8) had the typical severe deformity since birth without any evidence of rickets. All the common characteristics previously mentioned, were present.

Her sudden death provided an opportunity to establish the relationship between the abnormal diaphragm and the anterior chest wall. Nothing characteristic was seen in the bony and cartilaginous components of the anterior chest wall. This served to prove that, in infants, the deformity is only created during inspiration by the pull and contraction of the diaphragm and that the bony and cartilaginous changes take place when the anterior chest wall has become more rigid during childhood and the deformity has assumed its characteristic contour. The elastic recoil of this infant's soft anterior chest wall after its last "breath" obliterated the deformity almost entirely. However, examination of the diaphragm disclosed an abnormal anatomic pattern. Instead of the normal trefoil membranous portion with the characteristic circumferential musculature, the membranous portion was found to be V shaped with its fibrous apex extending to the gladiolar-xiphoid junction. Both limbs of the V were very narrow while the lateral muscular portions were unusually large and muscular. The anterior muscular portion attached to the sixth cartilages was comparatively small with poorly developed musculature. Contraction of this diaphragm, particularly its strong lateral portions, created a diffuse pull dorsally on its anterior attachments, namely, the sixth costal cartilages as well as the gladiolar-xiphoid junction. This created the inspiratory retraction of the lower anterior chest wall and the horizontal grooves (Harrison's). These findings were confirmed in one other infant. The question now arises, how does this diaphragm differ from the diaphragm that will create a chondrosternal depression (funnel chest)? The only

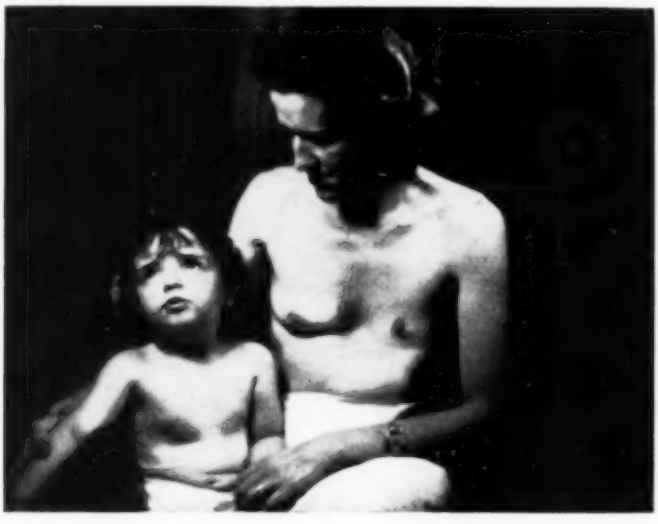

FIGURE 10: Mother with congenital chondrosternal depression and congenital absence of left breast. Child normal except for pronounced congenital chondrocostal grooves (Harrison's) present since birth. This demonstrates the hereditary as well as congenital tendency.

evidence[1] available is the diaphragm found in the four week old suckling dog with a typical congenital chondrosternal depression. In this diaphragm, the anterior portion was lacking in muscle fibers and indicated an underdevelopment of the septum transversum. It was not a case of "a short central tendon." From this scanty post-mortem evidence but sufficient clinical evidence one may conclude that the mechanical action of the diaphragm resulting from uneven and abnormal muscular development, particularly its anterior portion, will produce an anterior chest wall deformity of varying degree and form. Any accompanying condition which increases the softness of the anterior chest wall or the depth of the inspirations will aggravate the deformity.

In order to better understand this deformity, one must correlate and distinguish the mobile deformity of the infant (Figure 8) and its development into the fixed characteristic deformity of the child and adult (Figure 9). In the infant, there is a diffuse inspiratory retraction of the lower anterior chest wall. The upper portion of the chest including the manubrium remains round and immobile. It is often accompanied by horizontal depressions which are commonly known as Harrison's grooves (Figure 8). This creates a relative prominence of the gladiolus of the sternum. This typical deformity is established with each inspiration but during expiration the normal elastic recoil of the anterior portion of the chest restores it to its normal position. As the infant grows from infancy into childhood, the thorax becomes more rigid and fixed and the deformity becomes stabilized into a very definite pattern. In older children and adults, the upper portion of the anterior chest which includes the manubrium and its adjoining ribs and cartilages, retains its infantile rotundity; whereas the lower portion of the chest with the gladiolus and the adjoining cartilages, are relatively retracted so that the lower portion appears elliptical (Figure 9). In the lateral view, the anterior contour of the chest assumes the shape of a question mark.

Congenital chondrosternal prominence in infants produces no symptoms in spite of the paradoxical movement of the anterior chest wall. In children and adults, there is practically no compression on the heart or lungs and therefore produces no cardiorespiratory symptoms. If such are present, they are due to a psychoneurosis or some other organic lesion. It is the author's opinion that surgical treatment is too extensive for only a cosmetic improvement of the fixed deformity of children and adults; but a phrenosternolysis would be justified in an infant with a mobile deformity of this type to prevent or diminish the inevitable fixed deformity which would otherwise develop.

Congenital Chondrocostal (Harrison's) Grooves

This interesting deformity of the anterior chest wall which was described over 100 years ago consists of horizontal depressions, the deepest portion of which are along the sixth costal cartilages. This cartilage is the costal origin and attachment of the major portion of the anterior segment of the diaphragm. Just as for congenital chondrosternal depression and congenital

chondrosternal prominence, many authors[7-10] have attributed various causes for its production. On critical analysis none of the causes or mechanisms so far described, satisfactorily or adequately explain the development of this deformity. No matter what cause has been suggested there are at least an equal number of exceptions and there are too many normal chests in the presence of the same "causal" conditions. The major source of confusion, has been the failure to observe and co-ordinate the mobile inspiratory creation of the deformity in the round chest of infants and its gradual evolution to the fixed depressed grooves in the elliptical chest of older children and adults (Figure 10).

If two large groups, infants and school children, are examined for chondrocostal grooves the incidence will be approximately the same. If only infants who have the characteristic distinct mobile inspiratory retraction during quiet respiration and children who have fixed grooves of 1 cm. or more depth are considered, it will be found that in the case of the infants practically none will furnish any clue as to the etiology. Among the school children with grooves, one may elicit a history of asthma, pneumonia, chronic bronchitis, enlarged tonsils and adenoids, each of which has been regarded as the cause of the condition. These illnesses are coincidental, but are regarded as the cause for the grooves, because the added dyspnea associated with these illnesses aggravates the deformity. Furthermore, this etiology would leave unexplained the presence of grooves in older children with otherwise negative physical findings and no history of any respiratory condition. Furthermore, both groups have manifested the deformity since birth and hence the same congenital etiologic factor must apply to both groups.

Let us compare the normal infant its bell-shaped immobile thorax and the infant with these grooves during quiet respiration. The normal diaphragm of the normal infant contracts its radiating circumferential musculature during inspiration without exerting any pull along its origin, the lower circumference of the thorax. If sudden increased diaphragmatic contractions occur, as in crying spells or hiccoughs, an increased pull is exerted along the entire origin of the diaphragm. However the weakest and hence the most yielding is the anterior segment of the diaphragm which is attached to the sixth costal cartilages almost perpendicularly with no costophrenic sinus. Thus the pull is affected at this point immediately. That is why in normal infants, during crying spells or hiccoughs, one can see the sudden transitory inspiratory retractions along the sixth costal cartilage identical to the so-called Harrison's grooves. In infants with this deformity, the retraction or groove occurs with each inspiration or contraction of the diaphragm during quiet respiration. The explanation for this phenomenon is a deficiency of muscular fibers in the anterior muscular segment that is attached to the sixth costal cartilage. When such a diaphragm contracts, the pull on the membranous portion of the diaphragm is transmitted to the weak anterior segment and pulls the sixth costal cartilage dorsally, thus creating the grooves. These grooves may be unilateral or bilateral and vary in depth from slight to severe depending

entirely on the distribution and deficiency of the musculature of this anterior segment. Such a diaphragm in a fourteen month infant with a congenital chondrosternal prominence accompanied by these grooves was described.[3]

It must be remembered also that in the thorax of the infant, the sixth rib is comparatively more horizontal and occupies a relatively higher position in the thorax. As the child develops, the sternum and anterior ribs descend a distance the length of two vertebrae so that in older children the grooves occupy a comparatively lower level. As the infant's chest becomes more rigid and fixed, the deformity becomes more correspondingly fixed at the point of maximum retraction. In the child and adult, therefore, we find those horizontal grooves fixed at a comparatively lower level but the deepest part of the depression remains at the sixth costal cartilage.

Naish and Wallis in their very comprehensive article[10] state "these results seem to support the suggestion that the most important factor in the production of Harrison's grooves is the deficient expansion of the lungs." However, if one follows a new-born infant with these grooves into childhood, one must reach the conclusion that the causative factor, whatever it is, must be congenital. Furthermore, the grooves are produced synchronously with the contraction of the diaphragm and can be eliminated on either side with the paralysis of the corresponding hemi-diaphragm by the injection of novocaine into the phrenic nerve on that side. Therefore there is a direct relationship between the creation of the deformity and the contraction of the diaphragm.

Naish and Wallis state in their article "they occur very frequently in children with the deformity known as pigeon breast, which is almost certainly due to severe rickets." Chondrocostal grooves occur frequently together with chondrosternal prominence and also with chondrosternal depression. The abnormal diaphragm of an infant with the former combination was described. There is no question that any condition which causes a softer bony chest wall, such as rickets, or any condition which increases inspiratory effort as in dyspnoeic states will increase the depth of the deformity. Since congenital chondrocostal grooves have all the common characteristics mentioned previously for congenital chondrosternal depression and chondrosternal prominence, then the diaphragmatic theory is applicable to all three deformities.

SUMMARY

The deformities of the anterior chest wall commonly known as funnel chest, pigeon breast and Harrison's grooves for which the author has suggested the terms congenital chondrosternal depression, congenital chondrosternal prominence and congenital chondrocostal grooves respectively, are discussed as a related group of congenital deformities of the anterior chest wall.

The present concepts of the etiology of these deformities, such as rickets, upper respiratory obstructions and severe cough and dyspnea are analyzed

and refuted. It is acknowledged that these conditions do aggravate the extent of these deformities.

Another concept is suggested to explain the mechanism and production of these deformities. In normal chests the normal muscular portion of the anterior division of the diaphragm contracts and pulls the anterior leaf of the trefoil membranous portion of the diaphragm toward the anterior chest wall which remains stationary. If the anterior portion is underdeveloped or has relatively less muscle fibers or is more tendinous it will be pulled dorsally by the rest of the musculature of the diaphragm and produce an inspiratory retraction of its anterior chest wall attachment. As the character and composition of this anterior portion of the diaphragm varies, so will the pull on the anterior chest wall. The variation in the amount and the site of the pull will correspondingly affect the degree of severity and type of each one of these three deformities.

RESUMEN

Se discuten las deformaciones de la pared anterior del pecho comunmente llamadas tórax en embudo, tórax en pecho de pichón, y muescas de Harrison para las que el autor sugiere los nombres de depresión condroesternal congénita, prominencia condroesternal congénita y muescas condrocostales congínitas respectivamente, considerándolas como un grupo de deformaciones congénitas de la pared-anterior.

Se analizan y se refutan los conceptos actuales etiológicos tales como el raquitismo, obstrucciones respiratorias superiores, tos severa y disnea. Se admite que estas condiciones agravan la extensión de las deformaciones.

Otro concepto se sugiere para explicar el mecanismo y la producción de estas deformaciones. En el tórax normal, la parte muscular anterior del diafragma se contrae y tira de la hoja anterior del trébol membranoso del diafragma hacia la pared anterior del tórax que permanece estacionaria. Si la porción anterior está mal desarrollada o tiene menos fibras musculares o es más tendinosa, será tirada hacia atrás por el resto de la musculatura, del diafragma y produce una retracción inspiratoria de su inserción anterior a la pared del tórax. Así como varía el carácter y la composición del diafragma, así variará la tracción sobre la pared anterior.

La variación en la magnitud y la ubicación de la tracción afectará correspondientemente el grado de severidad y el tipo de cada una de estas deformaciones.

RESUME

L'auteur met en discussion, dans le cadre des déformations congénitales de la paroi antérieure du thorax, celles de ces déformations que l'on a l'habitude de mentionner comme "thorax en entonnoir," "poitrine de pigeon" et "gouttière d'Harrison." Il propose pour les désigner les termes de dépressions chondrosternales congénitales, de proéminence chondrosternales congénitales, et de gouttières chondrosternales congénitales selon les cas.

Il analyse et réfute les conceptions actuelles au sujet de l'étiologie de ces déformations, comme le rachitisme, l'obstruction des voies respiratoires

supérieures, la toux violente, et la dyspnée. Il admet que ces conditions aggravent le degré de ces difformités.

L'auteur suggère una autre explication du mécanisme et de la production de ces anomalies. Pour des thorax normaux, la portion musculaire de la division antérieure du diaphragme contracte et fait pression sur la feuille antérieure de la partie membraneuse trifoliée du diaphragme. Cette action s'exerce sur la partie antérieure du thorax qui reste fixée. Si cette région diaphragmatique est insuffisamment développée, ou à relativement moins de fibres musculaires, ou bien est plus tendineuse, elle sera entrainée en arrière par le reste de la musculature diaphragmatique. Ainsi se constituerait une rétraction inspiratoire des insertions thoraciques antérieures. L'action sur la paroi thoracique antérieure est fonction du caractére et de la composition de cette partie antérieure du diaphragme. Les variations dans la présence et la situation de la pression constitueraient les différents degrés de gravité et d'aspect anatomique de chacune des trois déformations étudiées.

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Heart Disease Control Program in the United States*

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About 9,200,000 people, or one in every 17 persons in the United States, has one or more of the cardiovascular-renal diseases, according to expert estimates.

Although we must estimate morbidity, we have more exact data on mortality, and as we examine it historically, a number of interesting facts come out. Figure 1 presents the crude death rates for the diseases that were the four leading causes of death in 1900: pneumonia and influenza; tuberculosis; diarrhea and enteritis; cardiovascular-renal diseases. The rates for the first three have shown a steady decline during the past half century, while those for the cardiovascular-renal diseases have increased. Infectious diseases are falling back under the pressure of better sanitation, better living conditions, increased public health measures, and the great new force of the antibiotics.

Figure 2 shows the per cent of decline during the past half century in death rates from seven diseases which are still important public health problems in some parts of the world. Even in 1900, these were not leading causes of death in the United States. In recent years none of them has accounted for more than two deaths per hundred thousand population.

As the infectious diseases have come under control, the average life span of our people has lengthened, and a larger proportion of the population is in the older age groups where the cardiovascular-renal diseases are more common (Figure 3). In 1950, more than 28 per cent of our population were 45 years of age and older, whereas in 1900 only about 18 per cent fell in the older age groups. It is interesting to see the effect of adjusting cardiovascular-renal disease death rates to the age of the population (Figure 4). When this is done, the total group shows rates that have been fairly level over the 50-year period. The cardiovascular diseases and, refining the classification further, diseases of the heart show some increase, although not so pronounced a rise as was indicated in the crude death rates.

The increase in the death rates from heart disease in the 1920's probably reflects better diagnosis rather than more heart disease. "Acute indigestion" began to be properly recognized as coronary thrombosis, after this common misdiagnosis was emphasized in 1918 by Tranter and Levine, in a paper entitled "Infarction of the Heart Simulating Acute Abdominal Conditions."†† The previous excellent report on coronary thrombosis written by Dr.

*Presented at the Second International Congress on Diseases of the Chest, Rio de Janeiro, Brazil, August 30, 1952.

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††Levine, S. A. and Tranter, C. L.: "Infarction of the Heart Simulating Acute Abdominal Conditions," *Am. J. Med. Sciences*, 155:57-66, 1918.

James Herrick in 1912 had not received much attention from the medical profession.

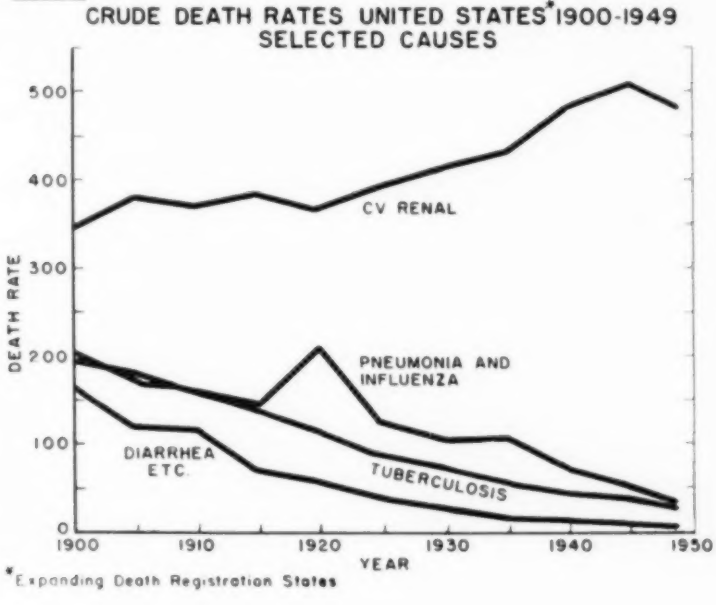
Another way of taking the measure of the problem is to see what proportion of all deaths has been due to cardiovascular-renal diseases (Figure 5). Responsible in 1900 for only one-fifth of all deaths, in 1949 they accounted for almost half.

If all deaths from heart disease and other circulatory diseases occurred in the elderly—if they were not preceded by long periods of disability—perhaps cardiovascular disease would not then justify the great efforts now being made against it in the United States. It is the death that occurs long before the Psalmist's allotted three-score years and ten (Figure 6) the increasing death rate in the productive years from 45 to 65, that especially concerns us. It is the loss to individuals and to the community, through prolonged illness from cardiovascular disease, that we are trying to prevent. In an aging population, measures to strengthen the health of people in middle and later life need special emphasis.

In 1922 a group of physicians who were interested in the increasingly great problem of heart disease formed the American Heart Association. Over the past 30 years, the efforts of this steadily growing organization have had an important effect on the care of patients with cardiovascular diseases. By setting up standards for heart clinics in hospitals, it has been instrumental in improving the quality of care offered. The Association has worked toward the use of standard nomenclature for cardiovascular-renal diseases and toward exact and careful diagnosis.

About four years ago, the Association underwent a reorganization which

Figure 1



changed it from a professional society to a broader voluntary agency with representation from the general public. Through this move, it has been possible not only to obtain support for important research programs but also to promote better public understanding of the problems and interest in dealing with them. Today the Heart Association is helping to counteract the idea that heart disease is hopeless and that little can be done about it. Its slogan is "New Hope for Hearts."

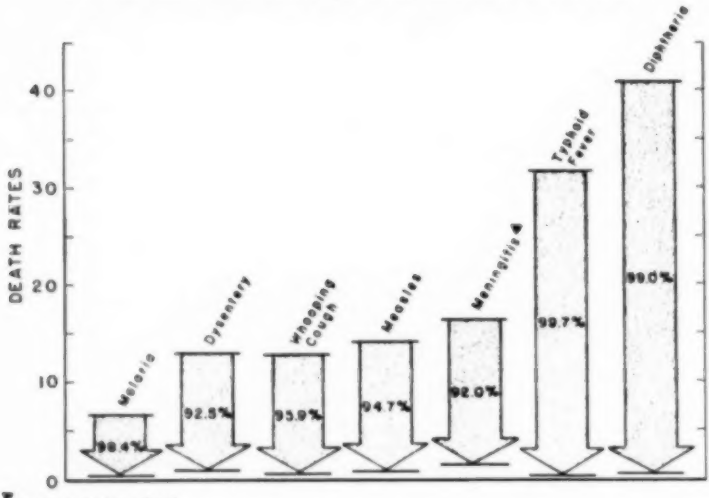
The first government program of national scope for the control of heart disease began in 1939. In that year, money for services to children with rheumatic fever and rheumatic heart disease was set aside from the appropriation for the crippled children's program of the federal Children's Bureau. Funds were allotted to the States to supplement their own resources for this purpose. Under this plan, a number of the States now provide services which may include diagnosis, treatment, hospitalization, convalescent care and continuing supervision for young rheumatic fever victims.

In more recent years, the Children's Bureau has also sponsored five regional surgical centers for children with congenital heart malformations amenable to surgery. By this means it is possible to give children in many areas the benefit of highly specialized surgery which formerly was available only to patients living near the large medical centers. Children whose condition is suitable for such procedures now are referred to the centers through the crippled children's agency of their own State, which also makes certain that they receive medical supervision after their return to their homes.

In 1948 the Federal Government took a second step toward controlling

Figure 2

DECLINE IN DEATH RATES FOR SELECTED CAUSES FROM 1900 TO 1949 IN THE UNITED STATES*



▼From 1909 to 1949
*Expanding Death Registration States

heart disease when Congress passed the National Heart Act. This Act established the National Heart Institute as one of the National Institutes of Health in the Public Health Service. It provided for federal support of research and training in diseases of the heart and circulatory system, and offered further aid to the States in the development of community programs for the control of these diseases. The National Advisory Heart Council was organized to advise upon the programs of the Heart Institute. It is made up of nationally recognized cardiologists and other prominent citizens; the Public Health Service, the Department of Defense and the Veterans Administration are also represented.

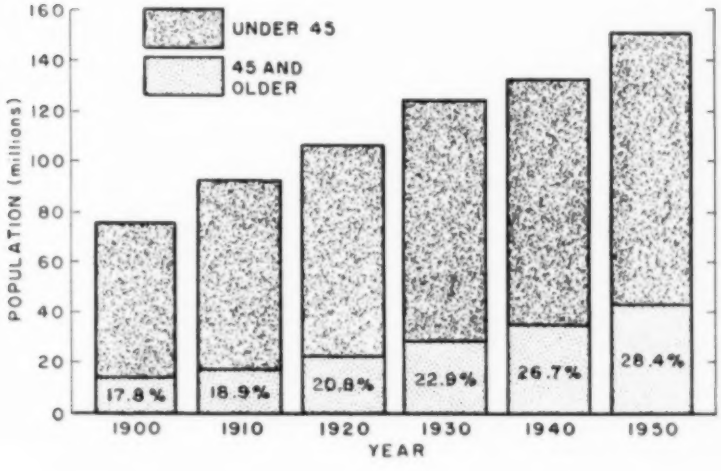
The National Heart Institute is now engaged in research aimed at increasing laboratory and clinical knowledge of the underlying disease processes affecting the heart and circulation. The search for new drugs and improvement of those already available are major interests, as are systematic studies in the field of hypertension. The field of arteriosclerosis is also receiving considerable attention; recent progress here has led to the development of new methods for the study of fat and cholesterol metabolism.

The Institute also makes grants-in-aid to individuals and institutions outside the Public Health Service for research projects and research training.

In the new Clinical Center of the Public Health Service which will open in July 1953, there will be about 100 beds for patients with cardiovascular-renal diseases. This is to be a research hospital, with twice as much space for laboratories as for patient care. Cases will be selected for intensive

Figure 3

POPULATION, UNITED STATES 1900-1950
AND PERCENT 45 YEARS AND OLDER



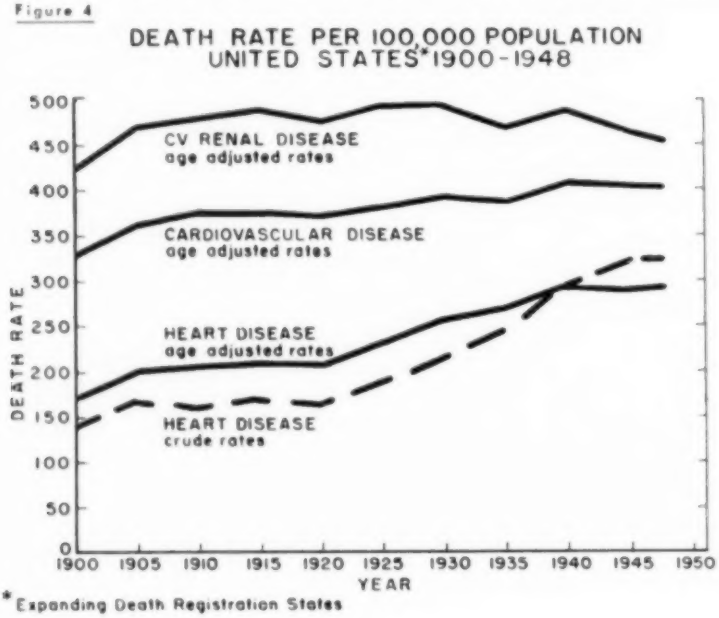
clinical study of certain disease problems which will be concurrently under study in the laboratory. It is hoped that the Center will contribute substantially to research in many fields, of which cardiovascular-renal disease is an important one.

The responsibility for encouragement of public health activities directed toward heart disease control in the States has been delegated by the Institute to the Division of Chronic Disease and Tuberculosis, which works together closely on programs of aid to the States. These include grants-in-aid as well as technical assistance, in the form of community demonstrations to develop and evaluate practical public health methods of heart disease control, and consultation to State and local health departments on those methods which offer promise.

These, of course, are not the only agencies which are concerned with the problem of cardiovascular disease. Medical schools all over the country are teaching the latest facts and techniques to a new generation of physicians and making major contributions to the body of knowledge about the heart and circulatory system in health and disease. State and local health departments are trying to develop methods of applying accepted knowledge in chronic disease control. Local voluntary associations give their assistance in many ways, from supporting research to providing services for cardiac patients. It is apparent, therefore, that while we have a great problem in controlling cardiovascular diseases, we also have constantly growing strength to deal with that problem.

A major line of attack on the cardiovascular-renal diseases is the expanding research program: basic research in physiology and chemistry,

Figure 4



*Expanding Death Registration States

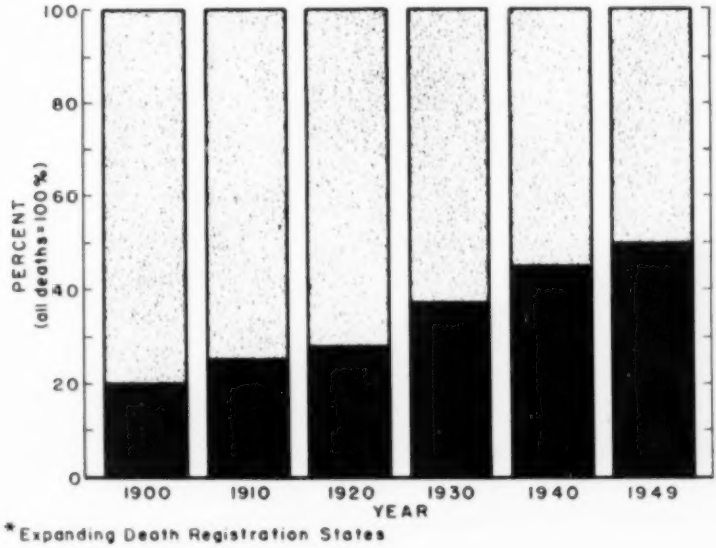
clinical, epidemiological, and biometric research—its scope and detail cannot be described here. Better understanding of the etiology of heart disease and improved therapeutic agents and techniques are gradually being acquired. But disease control programs have never waited for final answers. As soon as enough facts are available to give direction to public health efforts, prevention and control programs get under way.

Just as Koch's discovery of the tubercle bacillus in 1882 opened the way for the beginning of tuberculosis control, so have the identification of various kinds of cardiovascular disease and some understanding of their nature made possible the beginning of heart disease control. As a matter of fact, public health measures which prevent some types of heart disease have long been a part of accepted practice in the United States. Immunization programs for diphtheria have cut down diphtheritic heart disease; improved nutrition has lowered the incidence of beriberi; and, because of the vigorous campaign against venereal disease, syphilitic heart disease is becoming less common.

The relationship of streptococcal infection to rheumatic fever has been sufficiently demonstrated to justify action, and programs to prevent both primary attacks of rheumatic fever and recurrences which may result in heart damage are already being launched in many communities. In Newton, Massachusetts, for instance, the Public Health Service took part in a community heart disease control program which included a vigorous campaign against rheumatic fever. An attempt was made to find all persons who

Figure 5

PROPORTIONATE MORTALITY CV-RENAL DISEASES
UNITED STATES*, 1900-1949



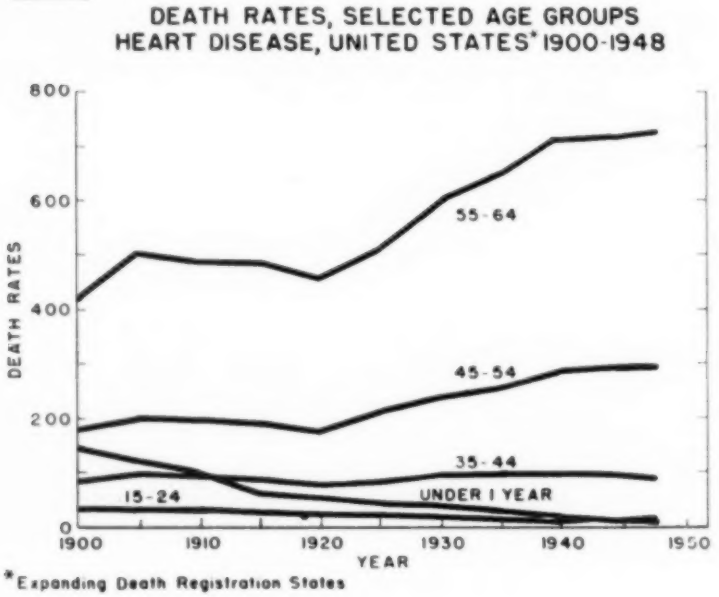
*Expanding Death Registration States

themselves had a history of rheumatic fever or in whose families there was history of the disease. The Health Department urged these people to see their physicians for advice on the prevention of streptococcal infection. Oral penicillin, the prophylactic agent accepted by the medical profession in Newton, was supplied at cost by the Health Department on the physician's prescription. The program was truly a community project, in which the Health Department, the medical profession, the schools and the hospitals joined forces.

Where we cannot prevent disease, we work toward the prevention of disability and of premature death. In heart disease, as in tuberculosis, early case finding helps to slow down progress of disease and to minimize disability. Even in arteriosclerotic heart disease or hypertension, where we lack specific preventive measures, much can be done to prevent complications. The importance of early detection of heart disease is therefore getting increasing recognition.

At present, there is no one instrument that is specific for case finding in heart disease as is the x-ray in tuberculosis. Even with its limitations, however, the chest x-ray is proving to be a valuable case finding device, since a considerable number of heart cases are found in chest x-ray surveys when films are read for that purpose. It is known, of course, that this procedure will not uncover many early forms of heart disease that do not produce cardiac enlargement. Also being studied for usefulness in screening for heart disease are the one-lead electrocardiograph, the sphygmomanometer and the ballistocardiograph. None of these alone, nor all of

Figure 6



them together, as yet give us a satisfactory screening device. When one is developed, community case-finding efforts will be more feasible.

There are certain aspects of the treatment of cardiac patients in which public health measures can support the private physician. One finds an excellent example in nutrition education. The patient with hypertension or congestive heart failure, whose physician prescribes a low sodium diet, needs rather extensive information and guidance in using it. Great success has been achieved with classes for such patients. This group teaching not only saves the physician's time but, in addition to teaching the patient what he should eat, gives the morale-building results of learning with a group. A booklet containing not only low sodium diet information, but also recipes and menus to help in preparing palatable meals, was developed by one health department with our help. It has now gone into its third printing, and physicians in many parts of the country are recommending its use by their patients.

One of the more hopeful methods of treatment of coronary heart disease has been the development of the anticoagulants. Their widespread use is hampered, however, by a lack of reliable, inexpensive laboratory techniques for their control. Physicians frequently do not use them because of the danger of hemorrhagic accidents, which can be avoided only if laboratory service is available for accurate and properly performed prothrombin tests. Last year the Public Health Service began to offer training in accepted methods of prothrombin time determinations to laboratory technicians in various parts of the country; thus far nearly 300 key technicians have attended the courses. They, in turn, are training others in the performance of the tests.

According to present day concepts, medical care does not stop with recovery of the patient from his acute illness but continues until he is restored to useful activities that are within his capacity. The measurement of work capacity, and, if necessary, training for employment within that work capacity, are a vital part of the prevention of disability and dependency from cardiovascular disease. At the present time we are planning such a project in cooperation with the federal Office of Vocational Rehabilitation, a medical school, a health department, and other local agencies. We will make objective measurements of eight-hour work tolerance of cardiac patients on the job, and of normal persons on the same job. These will be compared with a series of laboratory tests which have been devised to measure cardio-respiratory function and body economy. Comparisons should lead ultimately to an index by which a cardiac patient can be properly placed in industry. Energy requirements of various jobs will also be evaluated. It is hoped that this study will give us practical guides for employment of cardiac patients, so that they and their employers will no longer be troubled by the doubts and fears that result when a patient is told simply to "take it easy."

Professional education and interchange of knowledge and ideas are other important facets of control. Through the American Heart Association's annual Scientific Sessions, held in conjunction with the meetings of other

medical associations, vital information about recently discovered facts and new techniques in therapy can be exchanged and made available to the medical profession as a whole.

In addition to its grants to medical schools to help them provide better education in the cardiovascular-renal diseases, the National Heart Institute offers traineeships to young practicing physicians that allow them to improve their competency in the diagnosis, prevention and treatment of heart disease.

Another important aid to professional education is *The Heart Bulletin*, a bi-monthly non-profit magazine designed to bring practical information on heart disease to the general practitioner who is not a heart specialist. Health departments and heart associations in 35 States and territories have now subscribed to 58,000 copies of the Bulletin. The Public Health Service is also seeking other ways to make new knowledge about heart disease available to the practicing physician.

In the first century Seneca wrote of heart disease: "To have any other malady is to be sick; to have this is to be dying." Through most of history it has been terrifying and final. But within the lifetime of most of us much has been discovered about this ancient malady. Already we can say that to have it is not always to be dying. I think we can hope for the day when its deadly force will be controlled.

SUMMARY

1) According to expert estimates, about 9,200,000 people, or one in every 17 persons in the United States, has one or more of the cardiovascular-renal diseases. As death rates for the infectious diseases decline, those for the cardiovascular-renal diseases become more significant. In 1949, these diseases accounted for almost one-half of deaths in the United States, as opposed to one-fifth in 1900.

2) A number of agencies in the United States are especially concerned with heart disease. The American Heart Association, formed in 1922, encourages research, gives professional leadership and, recently, has organized lay volunteers in the campaign. The Children's Bureau has programs in rheumatic fever and heart surgery for children. The National Heart Institute of the Public Health Service supports and carries on research in basic physiology and chemistry, and in clinical, laboratory and biometric problems of heart disease, and the Division of Chronic Disease and Tuberculosis of the Public Health Service explores, demonstrates and evaluates public health methods of heart disease control.

3) Preventive measures have long been taken against diphtheria, malnutrition, and venereal disease, which sometimes result in heart disease. Now programs to prevent rheumatic heart disease through prevention of and early treatment of streptococcal infections are being launched.

4) Where we cannot prevent heart disease, public health programs can help prevent disability and premature death: early case-finding; support to private physicians in nutrition education in the use of low-sodium diets; training of technicians in prothrombin time determinations in use of anti-

coagulants; measurement of work capacity and re-training, and professional education are important facets of control.

RESUMEN

1) De acuerdo con los cálculos de los expertos aproximadamente nueve milliones doscientas mil personas o sea una de cada diecisiete personas, tiene una o más enfermedades cardio-renales. A medida que la proporción de muertes por enfermedades infecciosas desciende, aquellas causadas por enfermedades cardio-renales, se hacen más significantes. En 1949 estas últimas enfermedades, fueron la causa de la mitad de las defunciones, en tanto que en 1900 apenas eran la causa de un quinto del total.

2) Hay varias instituciones en los Estados Unidos que se preocupan por las enfermedades cardiacas. La American Heart Association, creada en 1922, impulsa la investigación, guía las actividades profesionales y recientemente ha organizado grupos de voluntarios no profesionistas en la campaña. La oficina de Niños, tiene planes en desarrollo sobre fiebre reumática y cirugía cardiaca en los niños. El National Heart Institute del Servicio de Salubridad Pública, sostiene y prosigue la investigación sobre fisiología y química básicas, así como sobre laboratorio, y problemas biométricos de las enfermedades cardiacas y la Sección de Enfermedades Crónicas y Tuberculosis del Servicio de Salubridad Pública explora, demuestra y valúa métodos de dominio de las enfermedades del corazón.

3) Se han tomado medidas preventivas desde hace tiempo contra la difteria, la desnutrición, y las enfermedades venéreas, las que algunas veces conducen a enfermedades del corazón. Ahora se han iniciado planes para prevenir la enfermedad cardiaca reumatismal, tratando de evitar y de tratar tempranamente las infecciones estreptocóccicas.

4) Cuando no se puede prevenir la enfermedad cardiaca, el programa de la Salubridad Pública puede ayudar para evitar la incapacidad resultante y la muerte prematura, por medio del descubrimiento temprano de la enfermedad, ayuda a los médicos particulares en la educación sobre la nutrición, y en el uso de las dietas pobres en sodio. También se preparan técnicos en determinaciones del tiempo de protrombina para el uso de anticoagulantes, medida de la capacidad de trabajo y rehabilitación, asé como la educación profesional que son aspectos importantes del dominio del problema.

RESUME

1) Les statistiques ont montré, après l'étude de 9,200,000 individus environ, qu'aux Etats-Unis, une personne sur 17 est atteinte d'une ou plusieurs affections rénales, ou cardiovasculaire. Alors que les statistiques montrent une chute de la mort par maladie infectieuse, la mortalité par atteinte cardio-vasculaire ou rénale devient plus évidente. En 1949, ces maladies représentaient environ la moitié des cas de morts dans les Etats-Unis, alors qu'elles ne représentaient qu'un cinquième en 1900.

2) Une certaine quantité d'organismes s'occupent spécialement des affections cardiaques. L'Association Américaine des Maladies du Coeur constituée en 1922, encourage la recherche, donne des directives professionneles, et a

organisé récemment une campagne faite par des personnes n'appartenant pas au milieu médical. Le Bureau d'Enfants s'occupe des rhumatismes articulaires aigus et de la chirurgie cardiaque infantile. L'Institut National du Coeur du Service Public de Santé aide et entreprend les recherches concernant la physiologie, la chimie et les problèmes de clinique, de laboratoire, et d'exploration fonctionnelle, propres aux affections cardiaques. La Division des Affections Chroniques et de la Tuberculose du Service Public de Santé fait l'exploration, la démonstration et l'évaluation de la valeur des méthodes d'hygiene sociale concernant la recherche des affection cardiaques.

3) Des mesures préventives existent depuis longtemps contre la diphtérie, la dénutrition et les affections vénériennes, qui parfois sont à l'origine des affections cardiaques. De nouveaux plans pour prévenir le rheumatisme articulaire aigu, et pratiquer le traitement précoce des affections streptococciques sont mis en route.

4) Quand il est impossible de prévenir l'atteinte cardiaque, l'hygiène publique est capable d'éviter l'incapacité physique et la mort précoce. Pour cela, il faut dépister l'affection à son début, il faut enseigner aux médecins praticiens des notions sur la nutrition et l'utilisation des régimes hypochlorurés. Il faut entrainer les techniciens à la recherche de la prothrombinémie pour utiliser les anticoagulants. Le dosage de la capacité de travail, et de la réadaptation ou du reclassement professionnel sont des éléments importants.

The Diagnosis and Management of Solitary Circumscribed Lesions of the Lungs

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Advances in diagnosis and therapy quite naturally stimulate interest in medical subjects and such is certainly true of the isolated pulmonary nodule. The more hopeful outlook justifies the placing of emphasis on this problem.

There is a trend toward pulmonary resection as a simple solution to the complex problem of solitary lesions of the lungs. The fact that a great many of such lesions should be treated by pulmonary resection does not diminish the physician's responsibility to those patients whose lesions should not be so treated; therefore, we should like to introduce this subject by emphasizing that the final decision regarding every patient with a solitary lesion of the lungs should be based on judicious individual consideration and not on a rule of thumb.

The great importance of this subject, the solitary pulmonary lesion, is well recognized by those of us interested in diseases of the chest. One of the chief reasons it is important is that some of the lesions are malignant. The proportion of circumscribed undiagnosed densities of the lung that represent malignant lesions is variously given as 20 to 55 per cent. Grow, Bradford and Mahon[1] in a series of 86 circumscribed, undiagnosed lesions, found 23 per cent to be malignant tumors and 49 per cent granulomas. Abeles and Ehrlich,[2] in New York City, explored 21 lesions, 33 per cent of which were found to be malignant and 20 per cent "tuberculomas." Watson[3] of Memorial Hospital found 40 per cent of 104 silent intrathoracic tumors to be malignant. Overholt[4] performed an exploratory operation on 145 patients for abnormalities discovered in roentgen-ray surveys and found that 24 per cent had malignant tumors. Harrington[5] reviewed a series of 52 cases of asymptomatic intrathoracic lesions in which he had carried out exploration and reported that 23 per cent represented malignant growths. Hood, Good, Clagett, and McDonald[6] recently studied 158 consecutive cases of solitary circumscribed lesions which were totally removed surgically. Of these lesions, 35.5 per cent were malignant, 16.5 per cent of the total series being primary bronchogenic carcinoma, 11.4 per cent metastatic lesions, and 7.6 per cent bronchial adenoma. Granulomas constituted 41.8 per cent of the entire group. Others have reported the total rate of malignancy for resected solitary pulmonary lesions as follows: Davis and Klepser,[7] 55 per cent; O'Brien, Tuttle and Ferkaney,[8] 42.9 per cent; Fink,[9] 33 per cent; and Sharp and Kinsella,[10] 27.3 per cent.

Although the foregoing figures do not represent comparable series of cases, the implication is the same for all of them, namely, that a signif-

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icant percentage of the clinically undiagnosable lesions of the lungs are malignant. It would seem unlikely, however, that as high a percentage of all solitary lesions of the lung are malignant as is represented by the figures just given. It is reasonable to assume that each of these series included cases in which there was a high degree of suspicion that the lesion was malignant, this being the reason for surgical removal and, therefore, inclusion in the report, whereas other cases were excluded by an equally high suspicion that they were benign. Too, malignant lesions are more apt to cause symptoms and therefore bring the patient to the physician, while benign lesions await accidental detection.

A second, and not insignificant, reason for this being an important subject is the high incidence of granulomas, as indicated by some of the foregoing figures. While many of the granulomatous lesions either heal or remain stationary, others break down, disseminate their contents and often spread the infection which has caused them. For this reason surgical extirpation is to be recommended for many such pulmonary lesions. Thus, it would seem that somewhere between 50 and 75 per cent of the solitary circumscribed lesions of the lungs are either granulomas or malignant tumors which should be removed. The remainder, not an insignificant number, are important because they cannot be distinguished from the above-mentioned lesions as a rule, and are treated by pulmonary resection because of the physician's inability to separate clinically the nonsurgical from the surgical lesions.

Difficulties in Diagnosis

Many factors are responsible for the difficulty in establishing the cause of most of these circumscribed lesions. Most such lesions are peripherally

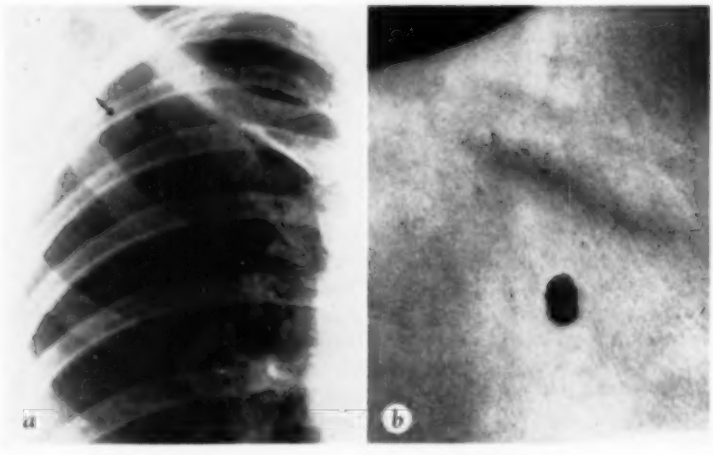

FIGURE 1A FIGURE 1B

Figure 1 (A and B): Pigmented mole causing a shadow that was thought to represent tuberculosis.

placed, beyond the limits of bronchoscopic visualization. In the case of tuberculomas, smears of sputum seldom demonstrate the organisms, and cultures of sputum and gastric contents for tubercle bacilli do not often give positive results. The long delay in obtaining the final culture report destroys the value of the procedure when one must act promptly because of the possibility that the lesion is malignant. Cultures for fungi take less than do cultures for tubercle bacilli, fortunately. The necessity of haste is a troublesome factor, yet prompt action is essential, for if the lesion is cancerous, delay of a few weeks may prove fatal. Malignant cells can be demonstrated in the sputum of only about 10 to 15 per cent of the cases of peripherally placed cancerous lesions, thus limiting the value of cytologic examination. The skin sensitivity tests for various pathogens are of limited value, as will be seen later. Roentgenographically, there are few distinguishing characteristics by which these shadows can be differentiated. In the majority of cases, these limitations are sufficient to prevent the making of a diagnosis except after surgical removal of the solitary nodule.

Intrathoracic Lesions Which Do Not Cause Circumscribed Shadows

Before discussing the lesions which do cause circumscribed solitary shadows we wish to mention a number of intrathoracic conditions which do not. Such shadows are not due to teratoids, dermoids, thymomas, intra-

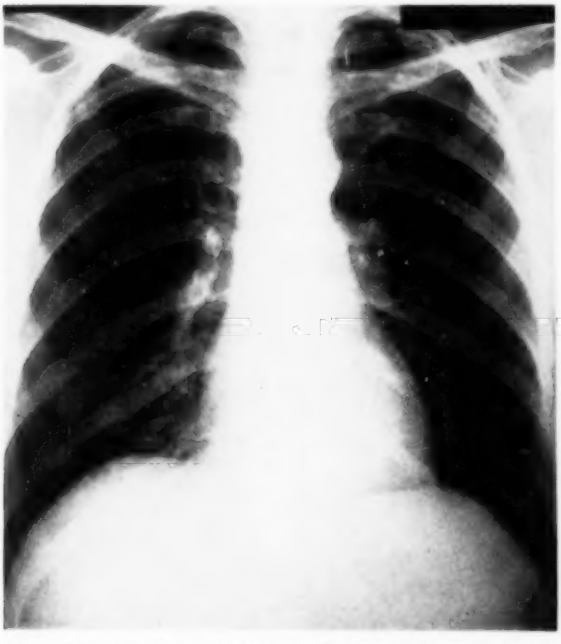

FIGURE 2: Unilateral shadow of the nipple resembling an intrapulmonary tumor.

thoracic thyroids, pleuropericardial cysts or diaphragmatic hernias and are rarely, if ever, due to arterial aneurysms or tumors of the lymphoma group.

Extrapulmonary Shadows Simulating Intrapulmonary Masses

The superimposition of roentgenographic shadows of lesions not actually within the lungs may resemble those of intrapulmonary masses and unless we localize every one of these lesions accurately, embarrassing errors in diagnosis will result. Lesions of the skin, such as tumors and moles, not uncommonly cast shadows that resemble nodules in the lung (Figure 1). The nipple shadow, when only one breast is present, may prove confusing (Figure 2). Tumors of the thoracic wall, such as osteochondromas of the

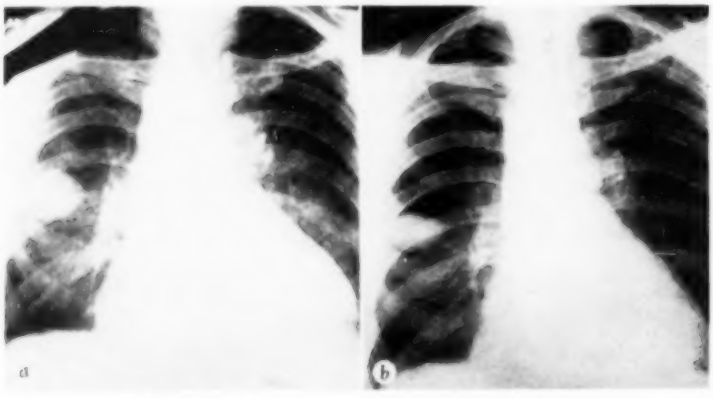

FIGURE 3A FIGURE 3B

Figure 3: Cardiac decompensation with interlobar pleural effusion. There was an interval of four days between A and B.

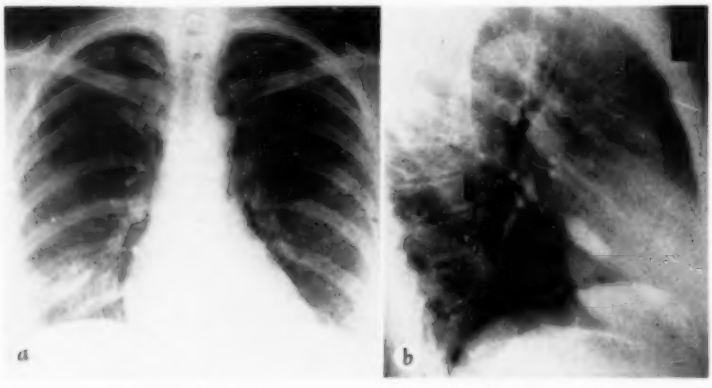

FIGURE 4A FIGURE 4B

Figure 4 (A and B): Fibrous mesothelioma of the interlobar pleura.

ribs or intercostal neurofibromas, have been interpreted as being intrapulmonary. Stereoscopic roentgenograms taken in the postero-anterior projection plus those taken in the lateral projection usually suffice to localize the process roentgenographically.

Certain pleural lesions such as encapsulated fluid (Figure 3) or empyema, fibrous mesothelioma (Figure 4), and pleural plaques may simulate lesions of the parenchyma of the lung, especially when they occur in the interlobar pleura.

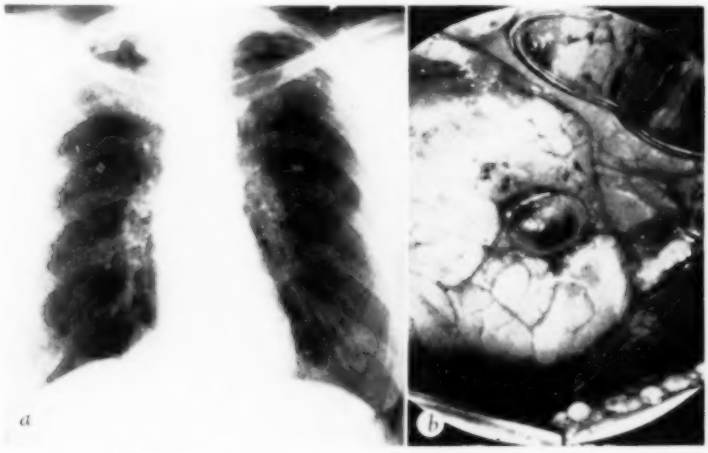

FIGURE 5A FIGURE 5B

Figure 5: Aneurysm of the inferior pulmonary vein. Circumscribed lesion discovered on routine survey. **A**, Roentgenographic appearance. **B**, Gross appearance at operation.

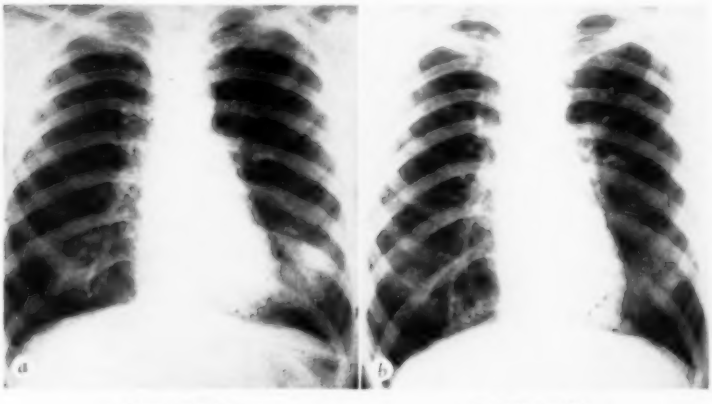

FIGURE 6A FIGURE 6B

Figure 6: **A**, Acute pneumonitis resembling tumor. **B**, Disappearance in ten days.

Pulmonary Lesions Causing Circumscribed Shadows

Vascular Abnormalities: Very few vascular lesions are solitary and circumscribed. Arterial aneurysms probably always are contiguous with the hilum and therefore are not discrete lesions. We have seen one aneurysm of the inferior pulmonary vein (Figure 5) which was a discrete lesion within the substance of the lung, but it is mentioned here only to emphasize the rarity of its occurrence.

Pneumonitis, Abscess, Infarction and Hydatid Disease: Acute pneumonitis may, on occasion, give a circumscribed density as seen on the roentgenogram (Figure 6). In such a case, however, the history of recent respiratory symptoms should lead one to repeat the roentgenographic examination after an interval, and in all likelihood there will be a definite change in the appearance of the lesion. Acute and chronic (Figure 7) abscesses of the lung often produce a discrete tumefactive lesion within the lung, and in such cases the history again should give the proper clue to their identity. Not to be overlooked is the common condition, pulmonary infarction (Figure 8), which may adopt a freakish nature and resemble an intrapulmonary neoplasm. Should such a situation occur and thoracic exploration be carried out, the patient would be subjected to a procedure performed under hazardous conditions and with no hope of benefit. Such cases emphasize the importance of the history and the need for individual consideration of every patient who has a pulmonary lesion. The case illustrated (Figure 8) also brings to mind the value of obtaining previously taken roentgenograms, which were available in this case and were of paramount importance in arriving at the correct diagnosis. On that rare occasion when one sees echinococcosis of the lung it is most likely to present as a well-defined, circumscribed mass, probably solitary.

Granulomas (Figures 9, 10, 11 and 12): As seen from the previous discussion, the granulomatous lesions are numerically the largest group under consideration here. They are clinically significant because of the pos-

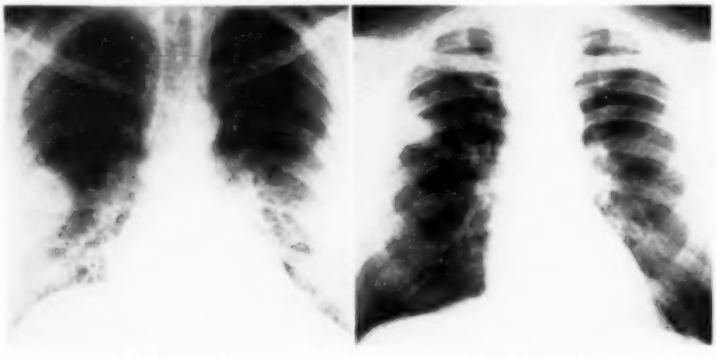

FIGURE 7 FIGURE 8

Figure 7: Chronic abscess of the lung.
Figure 8: Old pulmonary infarct. Review of previous roentgenograms made clinical diagnosis possible.

sibility of dissemination of the infection causing them. Pathologically, the different types of granuloma can be distinguished from each other only by identifying the causative organism or substance. From microscopic examination of the tissues identification is possible only in some cases of histoplasmosis, coccidioidomycosis and actinomycosis, and in oil granuloma. One cannot be sure of the identity of tubercle bacilli because they may be simulated by other acid-fast organisms and debris, and their identity must be confirmed by culture and guinea pig inoculation in any event. It is, therefore, a waste of time to search for acid-fast organisms in tissue sections.

Bacteriologic examination of granulomatous tissue removed at operation is fully as important as the microscopic examination of excised tumors and should consist of (1) microscopic examination of direct smears and fixed sections to search for and identify fungi and other causative agents, (2) culture for routinely encountered organisms, tubercle bacilli and fungi, and (3) guinea pig inoculation. If structures suggestive of tubercle bacilli or fungi are found in the histologic sections, the interpretation should be confirmed or corrected by cultures. Dr. L. A. Weed[11] of the Mayo Clinic conducted such a study of the specimens removed from 81 patients with granulomas of the lung and was able to identify tubercle bacilli in 13, Coccidioides in three, and Brucella suis, Streptococcus faecalis, Escherichia coli, Staphylococcus and Micrococcus in one case each. The remaining 60 failed to yield causative organisms. The cause of these 60 bacteriologically

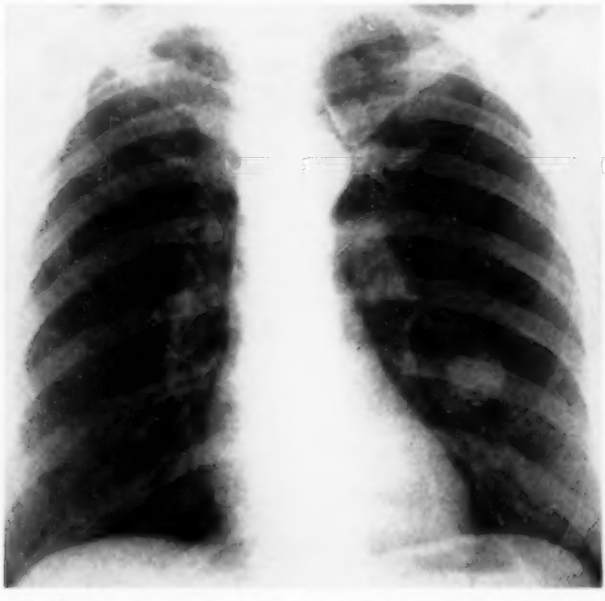

FIGURE 9: Tuberculoma. Tissue positive for tubercle bacilli on culture and guinea pig inoculation.

negative granulomas is unknown. However, many of these have been referred to as a Ghon tubercle in the past, a lesion which has been commonly regarded as the residuum of primary tuberculosis. Such a concept has a number of fallacies. These lesions are frequently inactive histologically (no giant cells or epithelioid cells) and it is likely that they do represent a "burned out" granulomatous infection. The nature of that infection is conjectural, however. We have seen "typical" Ghon tubercles which yielded

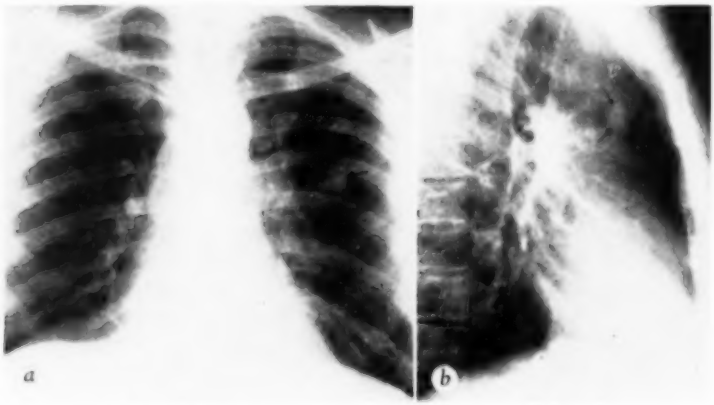

FIGURE 10A FIGURE 10B

Figure 10 (A and B): Granuloma. Tissue culture yielded Coccidioides immitis.

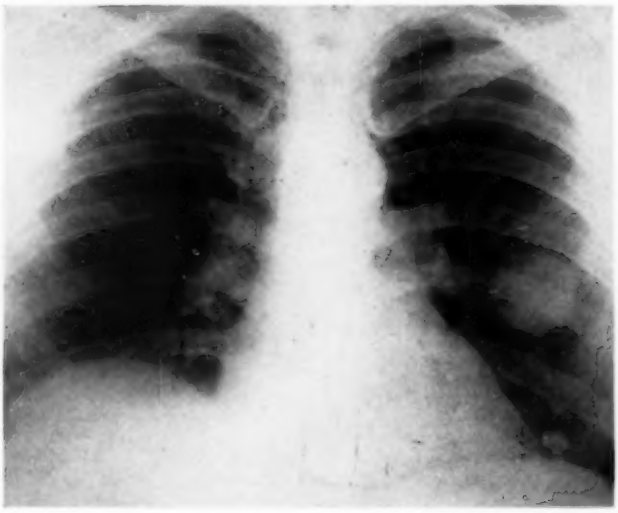

FIGURE 11: Granuloma removed surgically. Tissue culture was positive for Brucella suis.

a few colonies of Coccidioides immitis on culture. We believe that no granuloma in the lung should be regarded as tuberculous when bacteriologic proof is lacking, and we feel that the word "tuberculoma" should be reserved for those granulomas proved bacteriologically to be due to the tubercle bacillus

One should not conclude a discussion of granulomatous lesions of the lung without consideration of the specific skin tests and their value as diagnostic tools. The authors of one article stated that if a patient with a circumscribed pulmonary lesion had a positive skin reaction with coccidioidin or histoplasmin, they did not then recommend thoracic exploration. Their reasoning is difficult to understand, and we mention this here only to condemn it. The skin tests are very helpful diagnostic aids, but the

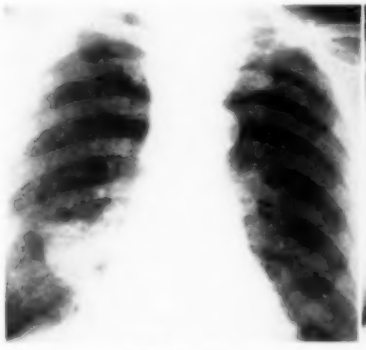

FIGURE 12

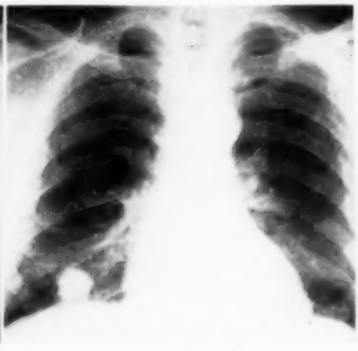

FIGURE 13

Figure 12: Oil granuloma, right lower lobe, proved at necropsy. The patient had ingested mineral oil for many years and the lesion had been present for eight years.—*Figure 13:* Hamartoma showing evidence of calcification.

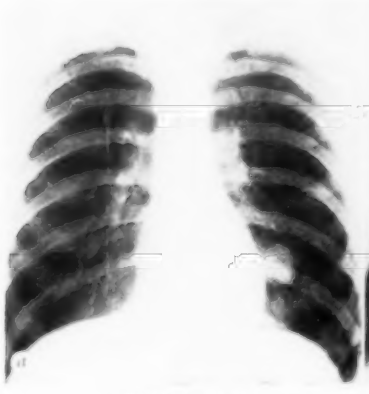

FIGURE 14A

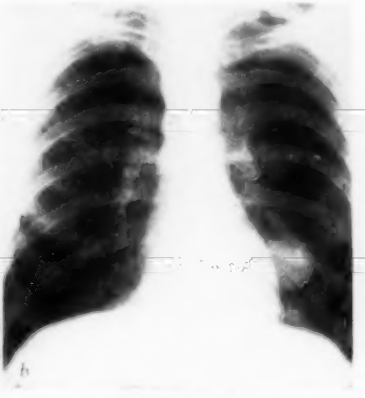

FIGURE 14B

Figure 14 (A and B): Bronchial adenoma showing increase in size over a four-year period.

results should never influence one in making the decision to explore or not to explore a thoracic lesion of unknown cause. It should be pointed out that if a localized lesion proves to be a manifestation of coccidioidomycosis or histoplasmosis, resection is good treatment. Among the multitude of people in the United States who are positive reactors to one or another of the specific antigens, a large number will ultimately have malignant lesions and the reaction to skin testing will not be altered or related in any way to such a lesion. The high degree of specificity of the tuberculin test is not yet substantiated for the other antigens.

Benign Tumors: Some of the peripheral lesions will prove to be benign tumors such as bronchogenic cysts or hamartomas (Figure 13). The former may on occasion undergo malignant changes, but the latter would never warrant resection if its absolute identity could be established preoperatively. Unfortunately, however, accurate identification of hamartomas is almost never possible until they are removed. They may even slowly enlarge while under observation, thus simulating other expanding types of tumors. When calcification is demonstrated within a hamartoma on roentgen-ray examination, as it frequently is, one cannot even then distinguish it from a calcifying granuloma. Thus, the majority of benign tumors will need removal in order to prove their innocence.

Adenoma of the Bronchus (Figure 14): When compared with other types of carcinoma of the lung, adenomas of the bronchus are relatively benign and definitely a distinct entity. Nevertheless, between 5 and 10 per cent of them will metastasize and therefore all of them are a menace and must be removed when found. As they are covered with mucous membrane they do not shed tissue and consequently no cells are present in the sputum. Most adenomas are centrally placed and within the range of bronchoscopic visualization and biopsy, but an occasional one will be peripheral, making

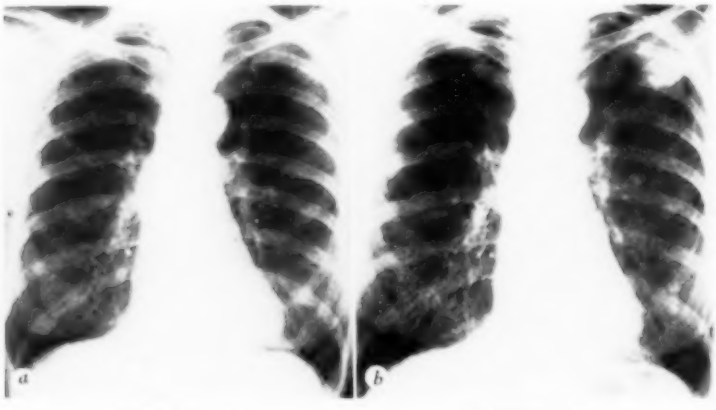

FIGURE 15A FIGURE 15B

Figure 15: The initial diagnosis was tuberculosis. After observation in a sanatorium the patient underwent left pneumonectomy for carcinoma. Death resulted from metastasis. Three years elapsed between A and B.

removal the only means of recognition. Moersch and McDonald[12] found that 91 per cent could be visualized bronchoscopically.

Malignant Tumors (Figure 15): The chief reason for concern and haste in the management of solitary lesions of the lung is the frequency with which they prove to be malignant and the disaster that befalls the patient when these tumors are not promptly recognized and removed. True enough, some of these solitary carcinomas are metastatic growths from tumors arising in other organs, but others are primary and curable and the two cannot be separated clinically. Malignant cells can be demonstrated in the sputum in only about 10 to 15 per cent of the cases of peripheral carcinoma, which is about the same percentage as is found in cases of metastatic lesions. Almost all the truly peripheral carcinomas are of the large cell or adenocarcinoma type.

There has been much discussion as to the size a nodule should be before it becomes clinically significant; this seems to be a rather hollow argument in view of the fact that every large, clinically important lesion was tiny during one period of its growth, and this would have been the time to eliminate it. The chance that a small nodule is significant is not as great as that of a large one, and yet none of them can be ruled insignificant on a basis of size alone.

If carcinoma is a possibility, one must not lose time with slow diagnostic methods such as cultures for tuberculosis. The practice of sending patients to the sanatorium for weeks or months in an effort to confirm a suspicion of tuberculosis must be abandoned. Unfortunately, many of our tuberculosis sanatoria are far removed from centers of population and are necessarily limited in their clinical and laboratory facilities for general diagnosis. Such institutions should not be considered diagnostic hospitals and patients should be sent to them only when a diagnosis of tuberculosis has been substantiated by acceptable procedures, but never when there remains a reasonable doubt.

The former practice of taking roentgenograms at intervals, waiting for some alternation in the size of a tumor, must also be condemned. Cancer of the lung may remain the same size for many months and to wait for some change may be the fatal delay. A "period of observation" is an obsolete tool in our diagnostic kit.

The factor of age as a guide to the diagnosis of carcinoma of the lung is proving to be unreliable as more and more malignant lesions are being recognized in younger people in their early thirties. Carcinoma of the lung occurs much more frequently in men than in women, but this difference is not sufficient to be of diagnostic significance.

Solitary Metastatic Lesions: The problem of solitary metastatic growths in the lungs raises a number of interesting and important questions. As mentioned above, it is rarely possible clinically to differentiate between primary and secondary cancerous lesions when dealing with solitary nodules, and in a rather high percentage of cases the pathologist may not be able to tell even after examining the surgical specimen whether a growth at its site of origin or merely a metastatic lesion has been removed. These

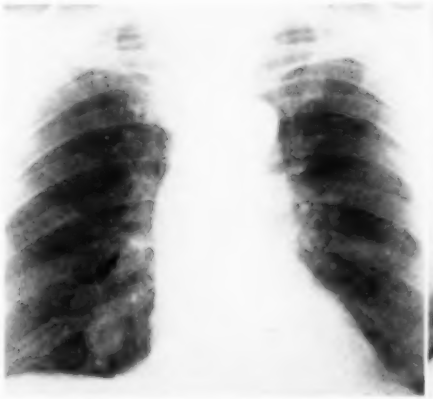

FIGURE 16

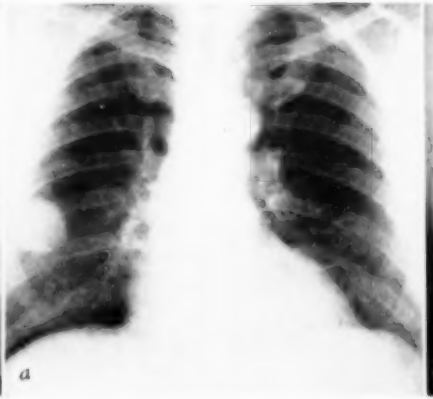

FIGURE 17A

FIGURE 17B

Figure 16: The patient underwent resection for an annular carcinoma of the rectosigmoid in 1922 and right hemicolectomy for adenocarcinoma in 1948, when the presence of a metastatic lesion in the lung was suspected. In 1951 resection of the lung for bronchial adenoma was performed.—*Figure 17 (A and B):* Granuloma. The value of tomography in demonstrating calcification is illustrated.

limitations to discernibility should be emphasized because the final results in any study of patients with such lesions will be considerably influenced by the number of patients with metastatic lesions included in the group—patients for whom there is no hope of ultimate cure.

When metastasis is a possibility one should make a reasonable search for the primary tumor. The word "reasonable" is used advisedly. The method and extent of any investigation to be conducted depend on the history and findings in the individual case and will vary accordingly. Surely any clue to the source of trouble should be carefully studied. On the other hand, one is not justified in going to extremes because the yield is poor when one is searching blindly for primary tumors and there are no symptoms or findings to point the way.

When a patient has or has had a known malignant condition and a solitary pulmonary lesion develops, we are not justified in jumping to the conclusion that the pulmonary affair represents metastasis of a malignant growth. It must be remembered that he is still subject to other unrelated disorders or may even have a second malignant tumor. When this situation prevails, individualization is of the utmost importance and many factors must be considered: the location and type of the first tumor, the extent and speed of its growth, its invasive characteristics, the time interval between removal or discovery of the original tumor and the appearance of the supposed metastatic tumor, the evidence of metastasis elsewhere, and the age and general condition of the patient. Under favorable conditions, exploration may be justified because (1) of the possibility that the pulmonary lesion may not be metastatic from the known original tumor (Figure 16) or (2) the removal of known metastatic tumors at times may be worth while as a palliative procedure. Obviously, the latter will be exceptional.

The Significance of Calcification

Before closing this discussion it is well to consider the significance of calcification within a pulmonary lesion. If the physician is to use this sign as a guide in the management of the lesion in question he must be absolutely sure that what he sees is calcium and that it lies within the mass. Often, tomography will demonstrate the presence of calcium in a lesion when it is not clearly shown on ordinary types of roentgenograms (Figure 17). Calcification may occur in a number of pulmonary conditions such as in old tuberculous foci, hematomas and hamartomas, around foreign bodies, within parasites, in thyroid and thymic tissue or tumors, in teratomas and dermoids, in granulomas, notably coccidioidomycosis and histoplasmosis, in pleural plaques, in metastatic osteogenic sarcoma, and perhaps rarely in a case of adenoma of the bronchus. Therefore, the presence of calcification is not specific. The presence of calcification indicates that the lesion is not a carcinoma, except in those rare instances in which a carcinoma has developed around a stone in the lung or as a secondary change in an otherwise benign condition such as teratoma. In spite of the comforting assurance that the presence of calcium gives, there are

some calcific lesions which should be removed. Granulomatous lesions may evacuate and spread the infection causing them. Many of them will yield positive cultures even though they may seem well calcified. Mahon and Forsee[13] have stated that the laminated or target lesions are always sterile, however. The calcification within a hamartoma or other neoplasm would not have the laminations seen in some granulomas. For the most part, lesions containing calcium may be left alone, but the clinician must exercise his judgment in determining those to be removed.

As seen from the foregoing, the problem of the solitary lesion of the lung is a complicated one imposing great responsibility on the physician. We cannot escape that responsibility by generalizations, but must individualize each case as it comes to us.

Note: Figures 7, 10A, 11, 12 and 17 are reproduced from Good, C. A., Clagett, O. T. and Weed, L. A.: "Granuloma of the Lung: A Problem of Differential Diagnosis," *Nat. Tuberc. A. Tr.*, 47:294-302, 1951.

SUMMARY

1) Solitary circumscribed lesions of the lung are significant because of the frequency with which they are malignant and the potential danger when granulomatous.

2) In the majority of cases the diagnostic limitations are sufficient to prevent making a diagnosis except after surgical removal.

3) Complete bacteriologic study of granulomatous tissue is as important as microscopic examination of tumor tissue.

4) The results of specific skin tests are of limited diagnostic value and are never conclusive in themselves.

5) The presence of a known extrapulmonary malignant lesion is not incontestable proof that an intrapulmonary nodule in the same patient is metastatic.

6) Slow diagnostic methods and prolonged periods of observation have no place in the management of the patient with a possible malignant tumor of the lung.

7) Most intrapulmonary nodules containing calcium need not be removed.

8) Careful individual consideration must be given each patient with a solitary lesion of the lung.

RESUMEN

1) Las lesiones circunscritas del pulmón son significativas por qué con frecuencia son maliganas y cuando son granulomatosas, potencialmente peligrosas.

2) En la mayoría de los casos las limitaciones del diagnóstico obligan a llegar a él sólo por la extirpación quirúrgica.

3) Un estudio bacteriológico completo del tejido granulomatoso es tan importante como el examen microscópico de tejido tumoral.

4) Los resultados de las pruebas cutáneas específicas son de limitado valor diagnóstico y por sí sólos nunca son concluyentes.

5) La presencia de una lesión malignan extrapulmonar comprobada no

es una prueba indiscutible de que un nódulo intrapulmonar en el mismo enfermo sea una metástasis.

6) No hay lugar para los métodos lentos de diagnóstico y los prolongados periodos de observación, al tratase a un enfermo de posible tumor maligno del pulmón.

7) La mayoría de los nódulos intrapulmonares que contienen calcio no necesitan ser extirpados.

8) Debe estudiarse cuidadosamente cada caso cuando hay una lesión solitaria del pulmón.

RESUME

1) Les lésions isolées circonscrites du poumon ont une valeur particulière à cause de la fréquence avec laquel le elles se montrent malignes, et leur danger en puissance lorsqu'elles sont granulomateuses.

2) Dans la plupart des cas, les possibilités de diagnostic sont telles qu'on peut le porter sans attendre l'extirpation chirurgicale.

3) L'étude bactériologique complète du tissu granulomateux a une importance aussi grande que l'examen macroscopique du tissu tumoral.

4) Les résultats des tests cutanés spécifiques sont de valeur limitée au point de vue diagnostique, et ne sont jamais suffisants à eux seuls.

5) L'existence d'une altération maligne extra-pulmonaire déjà connue n'est pas une preuve incontestable qu'il s'agit de métastases pour la masse intra-pulmonaire constatés chez le même malade.

6) Il n'y à pas place pour des méthodes de diagnostic lentes et de longues périodes d'observation quand il s'agit d'un malade chez lequel existe la possibilité d'un cancer du poumon.

7) La plupart des nodules intra-pulmonaires comprenant des parties calcifiées doivent être excisés. Une observation attentive est nécessaire pour chaque malade atteint d'une lésion isolée du poumon.

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Firefighting and Heart Disease

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Experimental and clinical evidence has accumulated in recent years supporting the view that certain strenuous occupations, such as firefighting, are capable of adversely affecting the cardiovascular apparatus to an appreciable degree. A fairly recent study of mortality by occupation among white male insurance policy holders shows that city firemen actually have the highest standardized relative index of mortality (125) for the principal cardiovascular renal diseases. While it has not always been possible to evaluate these disabilities equitably because of legislation based upon ancient views held in most states, sufficient evidence has been amassed in recent years suggesting the need for a wider acceptance of firefighting as an occupational factor in the production or aggravation of certain heart diseases.

Etiologic factors to be considered are environmental extremes (temperature and humidity), stresses and strains, trauma and shock, burns, and smokes and gases to which firemen are repeatedly subjected.

Stresses and Strains.

Several cardiovascular diseases currently considered to be more or less independent clinical entities may be related to stress and strain in one way or another. Among these are hypertension, arteriosclerosis and certain collagen diseases.

Expressed or repressed emotions or muscular effort may cause large rises in blood pressure. Hypertension may occur because one or more pressor mechanisms become unduly overactive in response to a systemic stressor. "Blast hypertension" has been found to persist for weeks in people who were in the vicinity of a major explosion such as the Texas City disaster. Frost and associates evaluated the effect of combined physical and mental stress on normal young healthy males. The "stress" in this study was the annual 500-mile Indianapolis Speedway Race. This is a competitive and dangerous contest in which the drivers race their cars around a two and one-half mile oval track at average speeds of between 120 and 125 miles per hour. There was evidence of increased pituitary adrenal stimulation during the stress period in that they showed marked decrease in the total number of circulating eosinophils after the race as well as an increased exertion of 17-ketosteroids of at least 50 per cent following the stress as compared to the control period. There is considerable clinical and experimental evidence that stress can also cause morphologic changes in the heart and that cardiac infarcts, hypertension and angina pectoris might be regarded as diseases of adaptation (stress).

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Hypertension, malignant nephrosclerosis and hyalinization with inflammatory arterial changes in the heart have been reproduced repeatedly by subjecting experimental animals to stress situations.

Numerous statistical surveys attest the fact that the incidence of arteriosclerosis appears to be definitely higher among individuals exposed to much stress and strain than in the population at large. The acceleration of arteriosclerosis with premature vascular breakdown is not uncommon as a result of the greater exposure of firemen and other individuals to hazardous physical tasks. It is a well known fact though inadequately explained that hypertension and arteriosclerosis enjoy a synergistic relationship since the presence of one so frequently favors the earlier production of the other. Such an acceleration of normal atheromatous changes may result in premature changes in the coronary and other arteries, terminating in an anginal syndrome or myocardial infarction. Rupture of the capillaries in an arteriosclerotic intima immediately following sudden strain and trauma, has been presented as evidence that they are possible precipitating factors in the development of coronary occlusion. The resulting hemorrhage may raise the plaque sufficiently to impede critically the coronary flow.

Forced or excessive muscular exercise so frequently experienced in firefighting and other strenuous occupations acts as the stressor agent and produces the alarm reaction of Selye. The goal of the circulation in exercise is to meet the enormous demands of the active muscles for oxygen and to help eliminate the carbon dioxide formed. Venous return is greatly increased with a markedly increased cardiac output. The output per beat (stroke volume) may reach 200 cc. and may be accompanied by a rise in blood pressure. It is obvious that the presence of underlying heart disease can be markedly affected by this change in cardiac dynamics which can disastrously affect a decreased cardiac reserve. This is especially true for those suffering from hypertension or coronary disease.

Numerous instances of myocardial infarction following great physical exertion are now on record. Anginal pain may be precipitated in patients with coronary sclerosis by induced anoxia. This phenomenon appears to be the consequence of both myocardial anoxia and the increased cardiac output and work due to lowered arterial blood saturation with oxygen.

Environmental Factors.

A. *Heat:* Exposure to high temperatures with the accompanying increase in the rate of circulation through the lungs and skin may lead to a more rapid absorption of harmful chemical substances from the lungs (such as carbon monoxide) or through the skin (such as coal tar derivatives). Studies made in various laboratories and in some industries have shown that the amount of physical effort decreases as the temperature increases. Indeed, at very high temperatures, especially when associated with high humidity, physical work may become impossible. Heavy work in high temperatures adds materially to the burden already placed on the body. The circulation must compensate for this excess heat at a time when there

is diminished heat loss due to the high temperature. Under these conditions, the body temperature tends to rise more rapidly and the strain on the circulation is great. The pulse rate increases while the stroke volume of the heart decreases markedly. When the heart attains its maximum rate, further work becomes impossible. Yet, firemen and others engaged in emergency work are frequently forced to labor in such environments. In "weather sensitive" individuals even moderate changes in atmospheric conditions can act as stress agents and produce somatic changes. The possible effects on a person with underlying heart disease, such as coronary sclerosis, are obvious. Focal myocardial degeneration with renal changes similar to those seen in the "crush" syndrome have been found regularly during pathologic observations.

B. *Cold:* Exposure to extreme cold likewise causes stress which affects most tissues of the body. Hyalinosis of the heart with muscular hypertrophy have been produced regularly in rats exposed to cold. Even fibrinous pericarditis and fibrin deposits within the heart have been found. Exposure to even moderate chilling causes an elevation in pulse rate and blood pressure often accompanied by various manifestations of vasospasm. In fact, continuous exposure to cold has been found to be a particularly effective type of stress in the experimental production of *persistent hypertension*, nephrosclerosis, cardiac hypertrophy and cardiovascular hyalinosis.

Trauma and Shock.

Following extensive traumatic injuries and shock, numerous cardiovascular phenomena occur. These include a fall in arterial blood pressure and decreased stroke volume owing to insufficient blood return to the heart. Morphologic changes affecting all organs include capillary damage, petechiae and edema. Degeneration of the myocardium may occur and reveals characteristic changes consisting of granular appearance of the fibers with basophilia, liquefaction and uneven density. Cloudy swelling and fatty degeneration of heart muscle fibers are conspicuous in patients who die of traumatic shock. Reports of coronary thrombosis following surgical shock are now on record. Similar changes are frequently noted following shock and collapse, especially in the presence of underlying heart disease. Subendocardial infarctions may be found owing to acute coronary insufficiency. Cardiovascular changes may also result from electric shock. When immediately nonfatal, a rise in blood pressure and various arrhythmias have been noted. Severe electrocardiographic changes have been recorded during ordinary electroshock therapy. Shock associated with "live wires" to which firemen and others may be exposed can result in similar damage.

Burns.

It has been recognized in recent years that extensive burns may also result in cardiovascular changes. Hemoconcentration and toxic absorption are important factors. Focal necrosis, calcification and even the

formation of granulomatous nodules have been observed in the hearts of rabbits. Some authors consider "serous myocarditis" with valvular edema to be quite characteristic of burns. It is often accompanied by panarteritis at a distance from the directly injured area, e.g., in the kidney. Less prominent degenerative lesions, such as cloudy swelling and fragmentation of the myocardial fibers have been noted in various species as well as in humans.

Smokes and Gases.

Atmospheric concentrations of smokes and gases to which firemen are repeatedly subjected may cause acute attacks of anoxia. They result in an increase in blood pressure and other vasomotor phenomena. The physiologic action of these toxic substances is associated with an interference with oxidation-reduction processes in the body cells. It is interesting to note that other agents to which firemen are repeatedly subjected may act in a similar manner. They include marked exertion and exposure to extremes of temperature which have been discussed previously. In dogs exposed to acute anoxia, cardiac edema and hemorrhages occur as a result of capillary damage. A severe oxygen-want of this type causes a generalized increase in capillary permeability with a predominance of symptoms and findings in the lungs (hemorrhage and edema) rather than the heart. In the presence of incipient heart disease this may precipitate cardiac infarction or failure.

A combination of anoxia and emotional stimulus, such as may occur during smoke exposure, are simultaneously applicable to many firemen and it has been shown that they act synergistically in the production of morphologic changes. The importance of anoxia in relation to the size of the oxygen debt is illustrated by the observation that normal individuals breathing air containing low concentrations of oxygen have increased oxygen debts after work.

Hazards from the following smokes and gases are especially applicable to firemen. Intoxications from various burning or escaped chemical compounds may cause manifestations of stress as well as poisoning. In addition to carbon monoxide which is generated by burning materials even in the presence of an excess of air, other irritating and lethal gases form which are synergistic in action. These include carbon dioxide, hydrocyanic acid, ammonia, hydrogen sulfide and sulfer dioxide. They are formed in the presence of burning rubber, silk, wool and many other organic substances. The presence of carbon dioxide induces increased respirations and more rapid death. Escaped gases from refrigerating and other industrial systems may also produce toxic changes (e.g., ammonia, formaldehyde, carbon tetrachloride, methyl chloride and other refrigerants). Carbon monoxide has an affinity for hemoglobin which is up to 300 times greater than for oxygen. This common gas produces an oxygen-want in tissues, which in the presence of underlying heart disease, may proceed to myocardial infarction. Chronic exposure to low concentrations for long periods may also produce permanent injury. Unfortunately, many of these

cases are returned to duty following resuscitation and a careful search for cardiac damage is seldom performed.

Symptoms and signs specifically related to the cardiovascular system include fatigue, dizziness, palpitations, dyspnea on slight exertion and precordial pain. Early exposure to low concentrations causes a rise in the diastolic blood pressure and rapid heart rate. Eventually, the systolic blood pressure may become elevated. This depressant action may lead to circulatory collapse as a result of marked diminution in muscle tonus and a failure of the venous blood to return to adequate quantity to the right heart. In man and experimental animals, this sequence has been followed electrocardiographically to the very moment of collapse and two findings are almost always noted: (1) a progressive diminution in the height of all T waves, beginning at oxygen concentrations of around 14 per cent and (2) a moderate depression of the ST segments. This objective evidence reflects the direct effect of anoxia upon the myocardium as well as changes due to the large outpouring of potassium from anoxemic tissues throughout the body.

SUMMARY

1) Hypertension, coronary thrombosis, the anginal syndrome, and manifestations of accelerated atheromatous changes are especially prone to occur in firemen and related dangerous occupations because of certain mental and physical factors associated with these occupations.

2) Adequate experimental and clinical evidence has been accumulated to show that the stresses and strains of firefighting, environmental extremes, trauma and shock, burns, and gases and smokes may act as predisposing factors in the causation of several cardiac disorders.

3) An aggravation of preexisting heart disease may also occur in the presence of the above factors.

RESUMEN

1) Debido a factores mentales y físicos que se asocian a la ocupación de los bomberos y otras actividades semejantes, la hipertensión, la trombosis coronaria, el síndrome anginoso y otras manifestaciones de cambios acelerados de la ateromatosis, ocurren mas a menudo en esas ocupaciones.

2) Se ha acumulado adecuada evidencia tanto experimental como clínicamente para demostrar que el esfuerzo y la tensión de la lucha contra los incendios, dificultades del ambiente, trauma, schock, quemaduras, gases y humos, son factores que actuan como predisponentes de varios trastornos cardiacos.

3) La agravación de la afección cardiaca preexistente puede ocurrir cuando se presentan estos factores.

RESUME

1) L'hypertension, la thrombose des artères coronaires l'angine de poitrine et d'autres manifestations dues à des altérations athéromateuses évolutives, ont tendance à frapper plus particulièrement les pompiers, et

semblent être en relation avec leurs dangereuses occupations. Il semble en effet que certains facteurs psychologiques et physiques en rapport avec leur activité soient en cause.

2) L'auteur a rassemblé des expériences concluantes et des constatations cliniques pour montrer que les secousses psychologiques, et la tension particulière de celui qui combat le feu, associées aux traumatismes, aux chocs, aux brulures, à l'aspiration de gaz et de fumées, peuvent constituer des facteurs prédisposants dans la constitution de différents désordes cardiaques.

3) Ces facteurs peuvent également être à l'origine de l'aggravation d'une affection cardiaque pré-existante.

An Evaluation of 95 Consecutive Pulmonary Resections for Tuberculosis*

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Introduction

The purpose of this paper was to evaluate the end results of 95 consecutive cases of pulmonary resection for tuberculosis done at the Veterans Administration Hospital, Livermore, California, during the period 1946 to 1951. There arose in our minds the possibility that thoracoplasty procedures were being supplanted by resections and for this reason a careful analysis of our resections might give us valuable information for future consideration. We found a steady decrease (Chart I) in the number of completed thoracoplasties done from 1948 which was a peak year for that procedure; while since 1947, there had been a steady increase in resections, with thoracoplasties and resections meeting at the crossroads in 1950.

Although from 1949 through 1951 there has been only a slight increase in the total number of resections done, a material decrease in the total number of completed thoracoplasties is observed.

An attempt is made to correlate our statistics with those already appearing in the literature. We have considered several phases in an attempt to bring more light on what we may expect in the future. These phases are an evaluation of the end results such as the number of patients well, patients sick, operative deaths, total deaths, complications, sensitivity and resistance studies of the organisms, and thoracoplasty procedures in relation to respective resections.

Patients referred to as being well are those who have had negative sputa plus stable x-ray shadows, who are working or are capable of working; in contrast to those who are referred to as sick who have positive sputa or changing x-ray shadows, who may or may not be hospitalized. Operative deaths are those that can be directly attributed to surgery and have occurred within 30 days of the date of surgery.

Material

Ninety-five consecutive pulmonary resections are reported. Seventy-eight patients are living of which 68 or 87 per cent have been contacted by

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follow-up examination or questionnaire. Of the entire series, 28 had total pneumonectomy while 62 had lobectomies and four were segmental resections.

The ages of those who had pneumonectomies varied from 25 to 64 years. The remainder varied from 24 to 57 years of age. The majority of the cases were white.

Results

In the 28 pneumonectomies, there were four surgical deaths. Acute cor pulmonale accounted for two of them; pneumonitis and irreversible shock accounted for one each (Chart II).

There were 15 (54 per cent) living and well while three (11 per cent) were still sick. Of the sick, one had a reactivation; one had a reactivation of endobronchial disease, and one remained with positive sputum from the time of surgery for a period of one and one-half years after which we lost contact with him.

In the 62 lobectomies (Chart III), there were 46 upper lobe, seven lower lobe, six bilobectomies, and three lobectomies plus segmental resections. Four cases were classified as segmental resections when in reality three were true segmental resections and one was a large caliber wedge resection.

There was one operative death due to right heart failure secondary to acute cor pulmonale and one death from transfusion reaction and blood

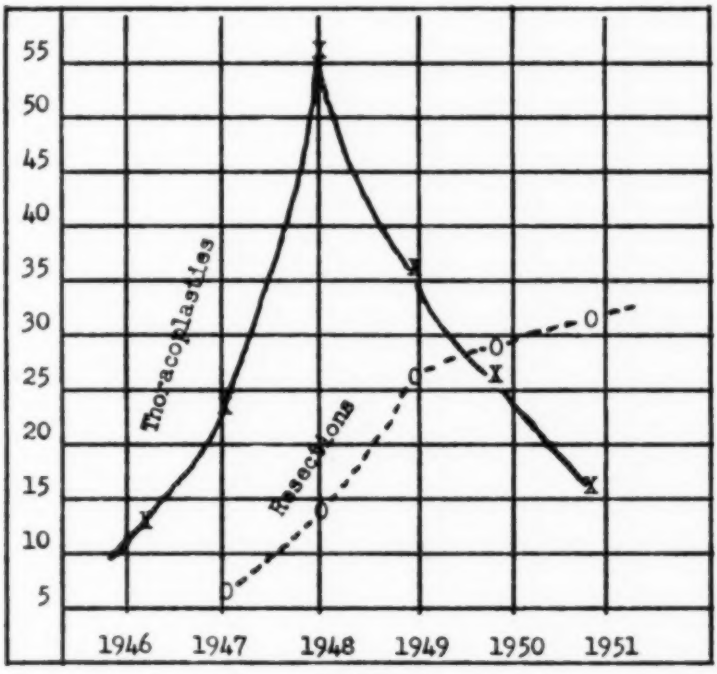

CHART I

diathesis of unknown cause. Massive intrapleural hemorrhage secondary to erosion of the pulmonary artery from delayed empyema occurring nine months after surgery accounted for one non-operative death; while two other deaths were cardiac. The fourth non-operative death resulted from a bronchopleural fistula with empyema of many months duration. Of the 62 lobectomies, 47 were living and well; while nine were still sick of which three had reactivations on the unoperated side. One had a spread, two had bronchopleural fistulas with empyema; one had empyema without fistula, and two had constantly positive sputa following surgery.

Three cases had segmental resection and one wedge resection. Of these, three are living and well; and one of the segmental resections is now dead following bronchopleural fistula and empyema with failure of expansion of the operated lobe. This patient had right upper and middle lobectomy six months prior to segmental resection on the left.

Complications

In the 28 pneumonectomies, there were two reactivations on the unoperated side; one empyema without demonstrable fistula and one bronchopleural fistula with empyema made a total of four direct complications (Chart IV). Both with reactivations are still living but sick. The one with empyema subsequently died as did the one with bronchopleural fistula.

A total of eight complications occurred in the 63 lobectomies. Six of

CHART II: PNEUMONECTOMIES

	Total	Well	Sick	Operative Deaths	Non-operative Deaths	Total Deaths
Number	28	15	3	4	6	10
Per cent		54	11	14	22	36

CHART III: LOBECTOMIES AND SEGMENTALS

Type	Total	Well	Sick	Operative Deaths	Non-operative Deaths	Total Deaths
Lobectomies	62	47	9	2	4	6
Segmentals	4	3	0	0	1	1

CHART IV: COMPLICATIONS

Type	No.	Broncho Pleural Fistula Empyema	Empyema Without Fistula	Spreads	Reactivations	Per cent
Pneumonectomies	28	1-D	1-D	0	2-L-S	14
Lobectomies	63	2-L-S 1-D	1-L-S	1-L-S	3-L-S	13
Segmentals	4	1-D	0	0	0	25
	95	Total Complications — 13				13.6

which followed upper lobectomies in which three cases had reactivation, two had bronchopleural fistulas with empyema, and one had an empyema without fistula. All of these are living but sick. The one death in this series of complications followed a bronchopleural fistula with empyema occurring after lobectomy with segmental resection.

There was one death in the four segmental resections. He developed bronchopleural fistula with empyema. No complications followed the seven lower lobectomies or the one middle lobectomy. A concomitant phrenic crush in four of the seven lower lobectomies did not appear to have any effect on the incidence of complications. In summary, there were 13 (13.6 per cent) complications in the 95 reported cases.

CHART V: SENSITIVITY AND RESISTANT STUDIES

	No.	Sensitive to 10 Mcg. Well	Sensitive to 10 Mcg. Sick or Dead	Resistant to 10 Mcg. and Sensitive to 100 Mcg. Well	Resistant to 10 Mcg. and Sensitive to 100 Mcg. Sick or Dead	Resistant to 100 Mcg. Well	Resistant to 100 Mcg. Sick or Dead
Pneumonectomies	14	2	4	4	0	1	3
Lobectomies	50	34	8	5	0	2	1
Segmentals	3	2	1	0	0	0	0
Total and Per cent		38/75	13/25	9/100		3/43	4/57

CHART VI: PNEUMONECTOMIES

	No.	Well	Sick or Complications	Operative Deaths	Total Deaths
Thoracoplasty Before	16	10	1	4	6
(Per cent)		63	6	25	38
No Thoracoplasty	6	1	2	0	4
(Per cent)		17	33		67
Thoracoplasty After	6	4	2	0	0
(Per cent)		67	33		

CHART VII: UPPER LOBECTOMIES

	No.	Well	Sick or Complications	Operative Deaths	Total Deaths
Thoracoplasty Before	12	7	3	1	2
(Per cent)		58	25	8	17
No Thoracoplasty	5	3	2	0	0
(Per cent)		60	40		
Thoracoplasty After	0	0	0	0	0
Concomitant Thoracoplasty	29	26	2	0	2
(Per cent)		90	7		7

Sensitivity and Resistance

Those cases which had sensitivity and resistance studies prior to resection were then evaluated. As of this writing, we have considered cases resistant to 100 micrograms of streptomycin as having a highly guarded prognosis. In the 20 pneumonectomies, 14 cases had such studies. Six were sensitive to 10 micrograms; and four were sensitive to 100 but resistant to 10 micrograms; while four were resistant to 100 micrograms (Chart V).

Both cases of reactivation were sensitive to 10 micrograms and of those that subsequently died, other than operative deaths, two were sensitive to 10 micrograms and three resistant to 100 micrograms.

A total of 50 lobectomies had preoperative studies of this kind of which 42 were sensitive to 10 micrograms. Seven of these were still sick and one dead. Five cases were sensitive to 100 micrograms and all were well. There were three cases resistant to 100 micrograms of which one was dead.

In the three segmental resections studied, all were found to be sensitive to 10 micrograms and of these one is dead.

CHART VIII: ALL OTHERS

	No.	Well	Sick or Complications	Operative Deaths	Total Deaths
Thoracoplasty Before	3	1	1	1	2
(Per cent)		33	33	33	66
No Thoracoplasty	1	1	0	0	0
(Per cent)		100			
Thoracoplasty After	2	2	0	0	0
(Per cent)		100			
Concomitant Thoracoplasty	2	1	1	0	0
(Per cent)		50	50		

CHART IX

	No.	Well	Sick or Complications	Operative Deaths	Total Deaths
Gale, Sweet, Day Chamberlain, Bailey, Himmelstein, Brantigan	745	496	95	47	137
(Per cent)		66.5	12.6	6.3	18.3
Glover	347	242			
(Per cent)		70	7.7		22
Davidson	200	151			
(Per cent)		75.5	4.5		20
Overholt	426	302		38	87
(Per cent)		70.9		8.9	20.4
Shabart, Samson	95	65	13	6	17
(Per cent)		68	14	6	18

Combining all resections, a total of 51 cases were found to be sensitive to 10 micrograms of which 38 (75 per cent), were well; and 13 (25 per cent), were sick or dead. Nine cases resistant to 10 but sensitive to 100 micrograms were all living and well. Three (43 per cent), of the total of seven cases resistant to 100 micrograms were well while four (57 per cent), were sick or dead.

Although we resected relatively fewer cases in the group resistant to 100 micrograms, we feel our end result of 57 per cent sick or dead is significant in that it points to a loss of operative patients in whom poor results can be expected. In 1950, J. C. Jones emphasized that streptomycin was the greatest aid in eliminating postoperative morbidity which has since been confirmed by others. He stated that every effort should be made to prevent the development of streptomycin resistance. Our statistics help bear out this opinion. When such resistant cases are selected for resection, we must expect an increase in morbidity and eventual mortality.

Grouping these sensitivity studies with the group of postoperative complications, it was found that nine of these cases with complications were sensitive to 10 micrograms; while in the group sensitive to 100 micrograms only one complication resulted, and the remaining ones occurred in the group resistant to 100 micrograms.

Thoracoplasty and Resection

An evaluation of the cases was made on the basis of their end results and whether thoracoplasty was done before, after, or concomitantly with resection or whether none at all was done (Chart VI).

In the 28 pneumonectomies, 16 patients had previous thoracoplasty of which 10 were living and well. One developed bronchopleural fistula and subsequently died. There were four surgical deaths and a total number of six dead. Six had no thoracoplasty, one of which is well and two had complications while four subsequently died. Of those having thoracoplasty after resection, four are well and two developed complications. No death has occurred in this group.

We were most anxious to evaluate this procedure in lobectomies, par-

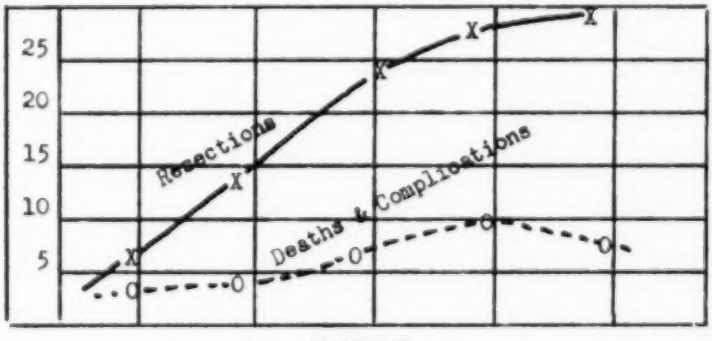

CHART X

ticularly upper lobectomies, where we have had the opinion that concomitant thoracoplasty can best be employed (Chart VII).

Twelve cases had thoracoplasty before resection of which seven are well and three had complications with one operative death and a total of two deaths. No thoracoplasty was done on five cases. Three patients are well and two complications occurred with those still sick. There were no cases in which thoracoplasty was done after resection. Twenty-nine cases had concomitant thoracoplasty and 26 are living and well. Two had complications. There were two deaths.

In the eight bilobar resections and lobectomies plus segmental resection (Chart VIII), there were three thoracoplasties done before resection of which one is well and one had complications. There was one operative death which made a total of two. There was one with no thoracoplasty who is well and two cases of thoracoplasty after resection both of whom are well. Two cases with concomitant thoracoplasty were done with one well and one who developed a complication.

Discussion

Such workers in this field as Gale, Sweet, Day, Chamberlain, Bailey, Himmelstein and Brantigan (Chart IX) reported their resections in 1949, 1950 and 1951 in which their combined efforts on 745 cases revealed 66.5 per cent well and 12.6 per cent still sick. The over-all operative mortality was 6.3 per cent with a total dead of 18.3 per cent. In 1950 Glover reported 347 cases with 22 per cent deaths with 269 living of whom 242 or 70 per cent were negative and 7.7 per cent positive for tubercle bacilli. The end result of our series of cases fits well into this general pattern with 65 (68 per cent) reported as well and 13 (14 per cent) still sick. There were 6 (6 per cent) operative deaths and a total number of 17 deaths (18 per cent). Overholt's recent reported series of 426 cases likewise compares to these reported series.

Of a total of six operative deaths, four occurred in the pneumonectomy group and it must be presumed that a more guarded outcome must be anticipated in this operation over the other resection procedures. On the basis of our statistics as well as those of others, it appears that lobectomy or segmental resection or any combination has a better chance of immediate survival from surgery. With 10 of the total dead of 17 occurring subsequent to pneumonectomy, it appears that this procedure is more hazardous even on a long term basis. With 54 per cent of the post pneumonectomy cases living and well against 76 per cent for all other resections, it appears that the lobectomy patient has again the advantage of ultimately getting well.

There were four (14 per cent) postoperative complications occurring following pneumonectomy while nine (14 per cent) followed all other resections combined. Our complication rate again compares to that reported by Clagett and Seybold in 1948 whose rates were 20 per cent for pneumonectomies and 28 per cent in lobectomies; or the report of Jones

in 1950 on 88 cases with 6.8 per cent complications; and finally those of Davidson in 1950 with a total of 21 per cent. Six of our nine complications, excluding pneumonectomies, occurred in the upper lobectomies. All of our reactivations are still alive; but the development of bronchopleural fistula with empyema appears to be a serious complication as all are dead but two and they are still sick. Empyema without fistula, although grave, carries only a slightly better prognosis.

Our study of sensitivity of the organisms to streptomycin produced interesting information in that 13 patients were sick or dead who were sensitive to 10 micrograms while none was sick or dead who was sensitive to 100 micrograms. Four of seven were sick or dead who were resistant to 100 micrograms. We would suspect on the basis of these figures that we no longer can accept a case who is sensitive to 10 micrograms as having the best prognostic factor to his advantage. Does this mean that if no other factors are ideally present that he stands the chance of a poor end result in spite of the fact that his organisms are sensitive to 10 micrograms? We do not believe this to be so, but are impressed with the fact that without these suitable factors complications can and will occur. What all the other factors might be we are not prepared to say. Those cases which are resistant certainly fall into the category in which the prognosis should be highly guarded. Whether the use of some of the newer drugs in these resistant cases will materially alter their eventual course following surgery cannot be stated at this time. We would like to suggest some factors being present which materially alter the course of these surgical cases. The outstanding one being the appearance of rather negative appearing bronchial mucosa as seen through the bronchoscope only to find at surgery, on dividing the bronchus, that gross evidence of disease exists in the submucosa and more peripheral tissue with rather normal appearing mucosa. This gross observation is often confirmed by microscopic study of the resected bronchus. Conversely, many times the mucosa and bronchus appear quite normal at bronchoscopy and surgery only to have the pathologist report bronchial tuberculosis. Can it be that antimicrobial agents although materially effecing mucosal involvement fails to possibly effect the lymphatic involvement within the wall proper? Rather than to attempt suture across such a grossly diseased area we feel that a more radical resection of the involved bronchus should be done even with plastic closure of same if necessary. There seems to be no question that inadvertently entering a cavity and causing gross contamination of the pleural space increases morbidity and mortality upon which the antimicrobial agents may have only limited value.

For the past two years we have been impressed with the use of concomitant thoracoplasty in resection. In this series none was done in the pneumonectomy group. This has been an actual survey of 95 consecutive cases and we offer no reason why concomitant thoracoplasty was not done in the pneumonectomy group but on the basis of our statistics and for reasons stated below, we now would advocate such a procedure. Our statistics showed that of those who had previous thoracoplasty 63 per cent were well and of those that had thoracoplasty after resection 67 per cent were

well; while those that had no thoracoplasty only 17 per cent were well. We feel that this speaks for thoracoplasty having a definite place associated with pneumonectomy. Thoracoplasty thus used with pneumonectomy tends to keep stabilized questionable active lesions in the other lung by preventing over distention. At the same time by preventing over distention, there is less chance of the patient later on becoming a respiratory cripple because of emphysematous changes in the remaining lung. This phenomenon was reported by Cournand and Berry; and Lester, Cournand and Riley as occurring particularly in the older age groups. For these reasons, although we report none, we feel a concomitant thoracoplasty with pneumonectomy has the advantage of eliminating another procedure which eventually becomes indicated. It has been our experience to find pneumonectomy much more difficult technically to perform under thoracoplasty and we suspect this may be reflected in our total death rate of 38 per cent over none in those cases in which the resection was done first.

We prefer to separate the lobectomies into two categories: (1) the upper lobectomies, and (2) bilobectomies and lobectomies with segmental resection. In the upper lobectomies who had concomitant thoracoplasty with resection there were 90 per cent well in contrast to 58 per cent well who had thoracoplasty done previous to resection; and 60 per cent well who had no thoracoplasty procedure. Ellis, Clagett and Carr in 1952 reported 14 lobectomies with concomitant thoracoplasty in which all are apparently controlled; while Conklin, Toby and Grismer in 1951 reported 38 cases in which 95 per cent were well. Woodington reported 20 lobectomies with 18 bacteriologically negative, one positive and one dead. In our series, complications developed in 40 per cent of those who had no thoracoplasty. In the combined procedure our total death rate was 7 per cent and we had no operative death. The deaths were of the late variety. We feel this is quite significant as some workers have signified a skeptical attitude towards the procedure because of a high death rate. It has been our experience that thoracoplasty immediately after resection is relatively non-shocking to the patient.

Recent reports on pulmonary resection for tuberculosis have shown better results with a lower postoperative morbidity and mortality. We have suspected for a long time that improved individual techniques along with wiser judgment in the selection of cases has played an important role in helping to improve the results. For this reason we attempted to correlate the number of resections done against the total number of deaths and complications on a so-called "Learning Chart." We think our chart bears out our individual improvement to some extent (Chart X). The discrepancy in the years 1949 and 1950 can be explained by the fact that in 1949 of the 24 cases resected 10 were pneumonectomies with seven being done in 1948 and four in 1947. This certainly made for more experience in this procedure; while in 1950 a greater campaign was made in the field of subtotal resections in which 22 of the 26 cases were of the subtotal type against four pneumonectomies, while in 1951, 24 varieties of subtotal resections were done against four pneumonectomies. There occurred only one surgical

death in pneumonectomies in these two years. Here again we feel we have become more efficient in the subtotal variety of resection.

SUMMARY

1) The number of patients in this series of 95 who subsequently became well compare favorably with those reported in the literature.

2) Complications post resection do not exceed those previously reported by other workers.

3) Patients sensitive to 10 micrograms of streptomycin should not be looked upon as having the better prognostic standard, but those resistant to 100 micrograms can be expected to have a higher percentage of increased postoperative morbidity.

4) Upper lobectomies with concomitant thoracoplasty gave a higher incidence of well patients and a lower incidence of complications.

5) Individual resection experience is another factor in improving the end results of pulmonary resection for tuberculosis.

RESUMEN

1) El número de enfermos que en esta serie de noventa y cinco mas tarde resultaron bien, se puede comparar favorablemente con los casos relatados en la literatura.

2) Las complicaciones después de la resección no exceden a las que antes han sido referidas por otros autores.

3) Los enfermos con sensibilidad de gérmenes a la estreptomicina a 10 microgramos no deben verse como los que tienen el mejor prognóstico, pero los resistentes a 100 microgramos puede esperarse que presenten el mayor porcentaje de morbilidad postoperatoria.

4) Las lobectomías superiores con toracoplastia dieron una frecuencia mayor de enfermos bien recuperados y menor frecuencia de complicaciones.

5) La experiencia individual en las resecciones es otro factor para mejorar los resultados finales de la resección pulmonar por tuberculosis.

RESUME

1) Dans ces séries de 95 cas, ceux qui furent ultérieurement stabilisés sont en proportion relativement favorable si l'on compare avec les cas publiés jusqu'à présent.

2) Les complications après l'exérèse n'ont pas dépassé celles qui ont été rapportées antérieurement par d'autres auteurs.

3) Les malades qui sont sensibles à 10 gammas de streptomycine ne peuvent pas être considérés comme ayant de ce fait un meilleur pronostic mais il est incontestable que ceux qui résistent à 100 gammas peuvent être considérés comme ayant un plus grand pourcentage de complications post-opératoires.

4) Les lobectomies supérieures avec thoracoplastie associée ont donné une plus grande proportion de stabilisations et un nombre moins élevé de complications.

5) L'expérience de l'opérateur est un facteur qui favorise les bons résultats dans les résections pulmonaires pour tuberculose.

The Use of Nebulized Trypsin
A Preliminary Report*

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Viscid bronchial secretions and exudates are important causes of disability in respiratory disease.[1-4] Liquefaction by nebulized proteolytic enzymes seemed a logical approach to this problem. The enzyme trypsin derived from the pancreas, and the enzymes streptokinase and streptodornase from streptococci have been shown to liquefy necrotic debris in selected cases of surgical wounds and in fibrinopurulent empyema[5-7] without causing injury to living tissues. Streptokinase is much less active in the presence of mucus than trypsin (Figure 1). Streptodornase is active on desoxyribonucleo-protein and desoxyribonucleic acid which are important viscous elements of purulent exudates.[2]

Preliminary in vitro studies on the sputum of a patient with acute bronchiolitis revealed trypsin's potentialities (Figure 1). Experience with tryptic† aerosol as a digestant of sputum in 17 patients with respiratory conditions is presented.

Procedure

A solution containing 40,000 to 50,000 Armour Units of specially prepared crystalline trypsin (Tryptar® Aerosol††) in 1 cc. of Sorensen's phosphate buffer solution (Tryptar Diluent) was placed in a vaponephrin apparatus and nebulized by 100 per cent oxygen flowing at the rate of 6 liters per minute. This solution was inhaled in 1 cc. doses three to six times daily, with an average complete course of treatment consisting of five doses. Following each inhalation, the patient was directed to rinse his mouth and gargle with warm water. Inhalations were started three to seven days after usual expectorants and bronchodilators had not produced desired effects on sputum. In most instances tryptic aerosol was administered during acute episodes occurring as complications of chronic pulmonary diseases. Diagnoses included pneumonia, bronchiolitis, bronchitis, bronchial asthma, bronchiectasis, chronic cystic lung disease, pulmonary emphysema and fibrosis (Table I). These patients in addition to receiving trypsin were given accepted therapy for their conditions. Five of the 17 patients received part of their therapy in the out-patient department; the remaining were hospitalized throughout the period of their treatments.

Sputums were collected every four hours starting one day prior to therapy and stopping 24 hours after the last inhalation. Each specimen was refrigerated until examined.

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*From the Cardio-Respiratory Division, Henry Ford Hospital, Detroit, Michigan.
**Deceased, June 15, 1953.
†Supplied through the courtesy of Marvin C. Ziporyn, M.D., of The Armour Lab.
††Tryptar Aerosol is a product of The Armour Laboratories.

TABLE I: RESPIRATORY CONDITIONS IN 17 PATIENTS* TREATED WITH TRYPTIC AEROSOL

Pneumonia	7
Bronchiolitis	7
Bronchitis	6
Bronchial asthma	2
Bronchiectasis	1
Chronic cystic lung disease, pulmonary emphysema and fibrosis	11

*Total number of conditions exceeds 17 since patients frequently had multiple conditions.

Results

Viscosity of Sputum: The sputum commenced to liquefy within four hours of institution of therapy. Digestion was evidenced by decreased viscosity, stringiness, color and sediment. Specimens collected three to six hours after the last dose of tryptic aerosol were 1.5 to 3 times as viscous as water. Sputa obtained prior to therapy were 10 to 20 times more viscous than water and frequently were not measurable by ordinary viscometers. Figure 2 depicts the effect of this therapy on viscosity of the sputum in a patient with status asthmaticus.

Of the 17 patients treated with tryptic aerosol, 10 had marked changes occurring in the fluidity of their sputums, two had moderate changes, one slight, two none, and two did not have sputum collections; the latter two showed clinical improvement. The two who had no changes occurring in their sputum had slowly resolving lobar pneumonia without expectoration prior to therapy (Table II).

TABLE II: EFFECT OF TRYPTIC AEROSOL ON FLUIDITY OF SPUTUM IN 17 PATIENTS

Marked increase	10
Moderate increase	2
Slight increase	1
Unknown (sputum not collected)	2
None (slowly resolving pneumonia, had no sputum)	2

Volume of Sputum: The volume of specimens usually increased during the first few hours following tryptic therapy and decreased afterwards. In a few instances, especially when longer courses of inhalation were given, the production of sputum ceased coincident with clinical improvement.

Clinical Course: Most of the patients were improved clinically, as noted by decreased dyspnea, wheezing, and rales. Constitutional improvement was also observed such as ability to sleep at night owing to relief from unproductive cough. Two with status asthmaticus were made more dyspneic during therapy although their sputums were influenced markedly (Table III). The seven with bronchiolitis received more benefit than those having lobar pneumonia,* bronchitis, asthma, and bronchiectasis.

Nebulized diluent (without trypsin) when given to two patients for one

*Had no sputum prior to or after tryptic therapy.

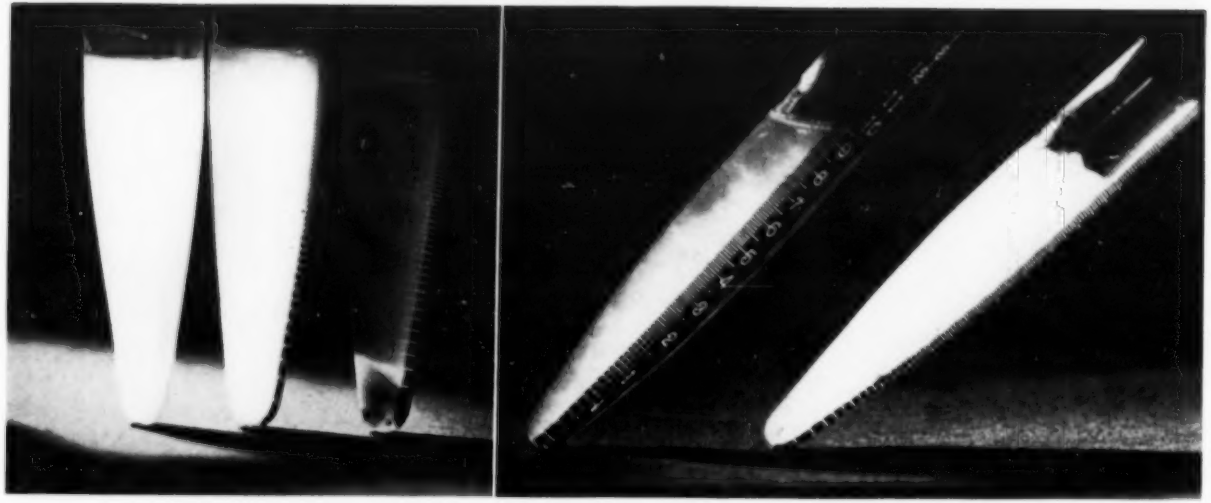

FIGURE 1 FIGURE 2

Figure 1: Trypsin diluent treated sputum (control) in left tube is semisolid; streptokinase-streptodornase treated sputum in center tube; tryptic treated sputum in right tube is approximately as viscous as water. All tubes contain same volume of sputum sample, and 0.2 cc. of added material. After standing 90 minutes at room temperature, the tubes were centrifuged.—*Figure 2:* Right tube contains patients' sputum before trypsin was given, and left tube contains sputum secured after three doses of tryptic aerosol. The change in fluidity is demonstrated by comparing the angles of the menisci with a horizontal line. Both specimens were centrifuged at the same speed for same period of time.

TABLE III: CLINICAL EFFECT OF TRYPTIC AEROSOL IN 17 PATIENTS

Helpful	11
Slight effect (viral pneumonia)	2
No effect (slowly resolving lobar pneumonia, no sputum)	2
Adverse effect (status asthmaticus, made more dyspneic)	2

day did not alter the sputums or clinical course. However, these two responded to tryptic aerosol. One with chronic bronchitis was relatively free of cough, wheezing, and expectoration for 10 to 12 days following a course of four tryptic inhalations. Over 50 per cent of the 17 were treated with the enzyme for one day only. It is possible that if longer courses of therapy had been given in this study, the number objectively improved would have been increased.

Patients' Personal Reactions: Thirteen of 17 patients stated the drug was helpful, five believed that therapy was mildly unpleasant, and four with moderate or severe dyspnea stated that the increased work of inhaling the medication was quite unpleasant.

Untoward Reactions

Untoward reactions consisted usually of mild local irritation of the oral and pharyngeal mucosa. Several with mild burning sensations or soreness of the tongue had a number of minute reddened areas on the lingual mucosa. One noted slight bleeding from a previously present shallow abrasion on the inner surface of the lower lip early in therapy. Bleeding did not recur when she rinsed her mouth well with warm water following succeeding inhalations. Another patient who did not rinse her mouth as directed had moderate oral and pharyngeal burning sensations lasting 24 hours after treatment had been discontinued. Transient hoarseness and brief loss of voice were observed in one patient (Table IV).

TABLE IV: SIDE EFFECTS OF TRYPTIC AEROSOL IN 17 PATIENTS

		DEGREE		
		Slight	Moderate	Marked
Local				
Burning or pinpoint erythema	8	6	1	1*
Transient hoarseness	1	-	-	1
Systemic	-	-	-	-

*Did not rinse mouth.

Contraindications: Recent gross hemoptysis, oral or pharqngeal lacerations, and severe dyspnea are the suggested contraindications to nebulized trypsin.

Indications

The suggested uses of nebulized trypsin include bronchiectasis, especially prior to bronchography; bronchiolitis and bronchitis; postoperative stelectasis; as an adjunct to therapeutic bronchoscopy or endobronchial catheterization; and in combination with other proteolytic enzymes to aid in digesting purulent elements. Undoubtedly other uses for tryptic aerosol will arise as studies proceed.

SUMMARY

Seventeen patients with respiratory diseases associated with dyspnea and viscous sputum and who were unresponsive to usual therapy were treated with tryptic aerosol. It was shown that tryptic aerosol decreased the viscosity of the sputum. Trypsin, promoting bronchial cleansing, was a helpful adjunct to usual therapy in the majority of these patients. Further evaluation is necessary before the full extent of the clinical usefulness of nebulized trypsin can be established.

RESUMEN

Se trataron con aerosol tripsico diecisiete enfermos de afecciones respiratorias caracterizadas por disnea y esputo viscoso que no habian mejorado con los tratamientos habituales. Se demostró que el aerosol tripsico hizo disminuir la viscosidad del esputo. La tripsina produciendo la limpieza bronquial, fué un adyvante util en la terapéutica habitual en la mayoria de los enfermos. Se necesita una evaluación ulterior antes de que pueda estimarse completamente la utilidad de la nebulización con tripsina.

RESUME

Dix-sept malades atteints d'affections respiratoires, accompagnées de dyspnée et d'expectoration visqueuse pour lesquelles il n'y avait aucune action des traitements habituels, furent soumis à l'action des aérosols de trypsine. Les auteurs démontrent que les aérosols de trypsine diminuent la viscosité de l'expectoration. La trypsine provoquant une évacuation du contenu bronchique réalisa une aide précieuse des thérapeutiques habituelles pour la majorité de ces malades. Des travaux ultérieurs sont nécessaires avant de pouvoir établir qu'elle est l'étendue de l'utilisation clinique de la trypsine en aérosols.

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Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis. A Critical Review*

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The introduction of oxygen or air in the peritoneal cavity for therapeutical purposes was used as early as 1893 in the treatment of tuberculous peritonitis. Its use in the treatment of pulmonary tuberculosis began in 1931 when Banyai, attempting to perform a haemostatic pneumothorax, injected air by accident in the abdominal cavity with resultant arrest of bleeding. With the publication of Banyai's papers in 1934,[1] the procedure has received special attention, and since then pneumoperitoneum has attracted wide interest as a method for pulmonary collapse.

The excellent book of Banyai published in 1946,[2] which contains a thorough study of the literature on artificial pneumoperitoneum, and many papers[3-17] have clarified the mechanism of its action, the indications and contraindications, etc., as well as the satisfactory results obtained in many patients with bilateral pulmonary tuberculosis, for whom no other form of collapse therapy or surgical procedure is indicated because of the extent of the disease.

The ever increasing use of pneumoperitoneum in the last decade, alone or associated with phrenic paralysis or modern chemotherapy for the group of desperate patients and the unexpected good results consistently obtained on many of such patients, deserve serious considerations. Even though it is frequently inadequate alone to produce arrest of the disease, it often produces sufficient improvement to permit major surgery, formerly forbidden.

The amount of air to be injected initially varies according to clinicians, usually from 500 to 600 cc. saves the patient much discomfort. Later insufflations of from 500 to 800 cc. are made at two or three day intervals until the desired degree of diaphragm elevation is obtained, which mainly depends upon the patient's adaptation, the rapidity of air absorption, and the objective to be attained.

The time required to obtain an intraperitoneal pressure sufficient to secure an adequate elevation of the diaphragm is from two to four weeks, depending mainly on the mobility and flaccidity of the diaphragm, the tonus of the abdominal muscles, the conditions of the lung and pleura, and the patient's adaptation. Following this period the amount of air can be increased and the interval between insufflations lengthened. Fluoroscopic control both before and after each refill is indispensable in order to determine the degree of the diaphragmatic elevation. Whenever possible, it is best to make insufflations of not over 800 cc. each seven to eight days. Intraperitoneal pressure following each refill should not exceed plus 10 cc. of water.

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Frequently in the beginning, pain is experienced in the shoulders (more often on the right side) and pain and oppression in the epigastric and hypochondrium.

Throughout the initial period the patient must be advised to remain in a supine position the greater part of the time and not to turn brusquely in his bed, to avoid unpleasant sensations. All of this disappears when the intraperitoneal pressure rises and the pneumoperitoneum is well established. If pain in the shoulders and the abdominal areas is intense, prompt relief results by raising the foot of the bed.

As an aid to pneumoperitoneum, the patient should wear a comfortable, well adjusted abdominal girdle, which aids in maintaining the intraperitoneal pressure and, consequently, diaphragmatic elevation. This enables many patients to tolerate the treatment in a better way and sometimes permits lengthening the interval between insufflations and reducing the amount of air injected.

Two points need to be emphasized in connection with the duration of the treatment, as follows: (1) In the final evaluation of each case, the individual peculiarities of the disease and the personal aspect of the problem must be taken into consideration. Treatment of so protean a disease as pulmonary tuberculosis cannot be subjected to hard and fast rules. (2) The limitations of the pneumoperitoneum as a method of pulmonary collapse therapy must be admitted, and the best way to check the effectiveness of the procedure is through the experience had with it by the physician applying it; when after an adequate period of observation, the expected results are not produced by the pneumoperitoneum, the treatment should be discontinued, since to extend it under such conditions defers the possible application of another type of treatment which might be more effective.

The first factor in determining the duration of the treatment is the degree of diaphragmatic elevation obtained, since to this factor is mainly attributable the therapeutic effectiveness of the procedure, and if the elevation of the diaphragm is inadequate following several weeks of treatment, pneumoperitoneum should be abandoned provided other treatment is feasible.*

The second factor to be considered is whether the pneumoperitoneum is used therapeutically alone for healing purposes or as a pro tempore treatment preliminary or preparatory to other types of surgical procedures. If the former, then the same principles as in treatment by artificial pneumothorax must be borne in mind, namely: (a) duration of the disease; (b) extent and localization of the pathological process; (c) presence or absence of cavities, their type, size and localization; (d) the predominant pathological character of the lesions, and (e) importance and extent of fibrosis. As with the use of artificial pneumothorax, it is good practice to continue a satisfactory pneumoperitoneum for not less than three to four years from the time it became effectual, that is, from the time the sputum

*In such cases, unless a special contraindication intervenes, there may be added to pneumoperitoneum, temporary phrenic paralysis on the side affected or worse, which in the majority of cases produces an adequate elevation of the diaphragm on that side.

was converted (gastric content, bronchial lavage, guinea pig inoculation).

The duration of pneumoperitoneum when employed as a preparatory method preliminary to another type of therapy, is conditioned on the results obtained. As soon as general and local conditions improve, the treatment should be suspended and followed by the selected surgical procedure.

Physiology and Mechanics of the Action

Essentially the action exerted by the pneumoperitoneum upon the lung does not differ from that exerted by the artificial pneumothorax, both of which are mainly efficacious by securing an adequate pulmonary relaxation and diminution of the volume of the lung.

The elevation of the diaphragm reduces the apico-basal diameter of the lung and the intrapleural pressure tends to become less negative, consequently reducing the pulmonary volume, which permits the elastic pulmonary structures to contract with resultant relaxation of the pulmonary tissue. In its mode of action, therefore, pneumoperitoneum differs from artificial pneumothorax in that the latter decreases the intrapleural negative pressure, thus permitting the lung to collapse, whereas pneumoperitoneum reduces the pulmonary volume by the elevation of the diaphragm, thus permitting the collapse of the diseased areas. It is then seen that, although the mechanics is different, the final result is the same, namely relaxation of the pulmonary tissue and diminution of the volume of the lung.

The impression is generally held that pneumoperitoneum is of special benefit in the treatment mainly of basal lesions; however, apical lesions respond favorably, which is accounted for by the fact that the intrapleural space totally encircling the lung is a closed cavity in which any change of the pressure will be equally distributed throughout the intrapleural and thoracic cavities and also by the fact that, because of the conical shape of the lung the changes of pressure proceeding from its base towards the apex should grow larger as they are transmitted to the apex.

Accidents and Complications

The wide acceptance of artificial pneumoperitoneum has increased progressively during the last decade and one of the arguments advanced to recommend it as a method of collapse therapy is the rarity of accidents and serious complications. Yet more than forty different complications and accidents may occur during its application.

Minor complications such as pain, anorexia and loss of weight at the beginning of treatment, nausea and vomiting, constipation or diarrhea, flatulence, hiccup, slight dyspnoea, etc., are generally of slight importance.

Air embolism is the most serious complication. Its symptomatology, prognosis, and treatment in no way differ from those found in air embolism from any other source. However, in pneumoperitoneum it can be virtually eliminated if a careful technique is employed.

The relatively frequent subcutaneous emphysema and the much rarer

preperitoneal emphysema and emphysema of the omentum are of no importance. Mediastinal emphysema is a rare accident which occurs when air passes from the peritoneal cavity to the mediastinum through the aortic and esophageal hiatuses and the hiatus of the vena cava. If the volume of air passing to the mediastinum is large, the patient will feel pain and oppression in the sternal and laryngeal areas, dysphagia, hoarseness, palpitations and pain at the shoulders sometimes radiating to the arms. In many cases, interstitial emphysema can be observed on the anterior surface of the neck and sometimes a tympanic area on both sides of the sternum. Although the condition is uncomfortable, it is not of great consequence and usually disappears in three or four days.

Tuberculous peritonitis occurs in approximately 1 per cent of cases. Nontuberculous intraperitoneal effusions are found with some frequency and are similar to the small sterile pleural effusions so frequently observed in artificial pneumothorax and which do not interfere with the treatment. Peritoneal adhesions occur with relative frequency but they are largely asymptomatic. Occasionally they produce intense pain or prevent the elevation of the diaphragm, which compels abandonment of the treatment.

Intraperitoneal hemorrhage is extremely rare but can be serious; it is produced either by the puncture of a blood vessel or by the rupture of a highly vascularized adhesion. Puncture of the abdominal viscera is rare and perhaps the accident occurs more frequently but cannot be recognized because it is entirely asymptomatic and without further consequence. Acute appendicitis is apparently more frequent in patients treated with pneumoperitoneum than in other tuberculous patients.

Subumbilical haematoma, scrotal and umbilical pneumoceles, torsion of the omentum or of the splenic pedicle, aggravation of hemorrhoids, rectal hemorrhage, retention of urine, amenorrhea and dysmenorrhea, cystocele, rectocele, and the development of hernias or aggravation of pre-existing hernias are sometimes observed. In older people and in those with cardiovascular disease, pneumoperitoneum may cause cardiac decompensation; displacement of the heart and the great vessels. Massive and smaller areas of pulmonary atelectasis have been found rarely during pneumoperitoneum treatment caused by bronchial kinking.

Artificial pneumoperitoneum has a wide field of indications in the treatment of pulmonary tuberculosis, but it should be clearly understood that it is in no way a competitor of other types of treatment

Present-day indications:

1) Bilateral lesions with or without cavity not too far advanced, where pneumothorax or other surgical procedures are not indicated. Pneumoperitoneum alone or combined with chemotherapy can provide a considerable improvement so that a unilateral pneumothorax or another surgical treatment may be done at a later date. Occasionally pneumoperitoneum is definitive in itself.

2) Relatively small lesions where bed rest alone or combined with modern chemotherapy are insufficient and in which pneumothorax could not be performed.

3) In pneumothorax complicated with pleurisy with persistent serofibrinous effusion after repeated aspirations and with the onset of empyema, attempt must be made by all means to obtain pulmonary re-expansion (large percentage of unexpandable lung). Pneumoperitoneum not only contributes to the re-expansion of the lung and obliteration of the pneumothorax space but also may be a partial substitute for the collapsing effect of pneumothorax.

4) In cases of haemoptysis uncontrollable by other means, when it is impossible to determine with certainty which lung is bleeding.

5) For pregnant tuberculous patients two or three days after delivery.

6) Cavitation of the lower lobes without evidence of uncontrolled endobronchial disease.

7) For patients under treatment by pneumothorax, thoracoplasty or pulmonary resection, when a secondary basal lesion develops in the same lung or on the opposite side. These cases usually respond well to pneumoperitoneum plus chemotherapy.

8) In patients with acute active and progressive tuberculosis who are too ill for immediate surgical procedure and in whom the risks of complications from pneumothorax are specially high; if temporary collapse therapy is indicated, pneumoperitoneum is exempt of serious complications and often produces sufficient improvement to prepare for other type of treatment. If necessary it can be safely discontinued. Recently chemotherapy is applied for the same purpose with good results and it is advantageous to use simultaneously in combination with pneumoperitoneum.

9) In cases of bronchial tuberculosis, if establishment of a reversible method of collapse therapy is required in order to control the paranchymal disease, pneumoperitoneum tends in some way to facilitate drainage and besides, if necessary, can be abandoned, which is not always the case with pneumothorax (unexpandable lung).

10) When collapse therapy is indicated and any other procedure has been found impossible or inadequate, pneumoperitoneum is the first step in a surgical program of collapse therapy. New chemotherapy has been added to pneumoperitoneum or has replaced it.

Contraindications

Contraindications are as follows:

1) Generalized tuberculosis, moribund patients, extreme toxicity.

2) Cardiac decompensation, deficiency of coronary circulation.

3) Plastic peritonitis, large abdominal tumor, large hernias of the abdominal wall.

4) Chronic fibroid disease with thick-walled cavities.

5) Other associated pulmonary diseases: silicosis, carcinoma, pulmonary suppurations.

6) Fixed diaphragm. When fluoroscopic examination reveals a fixed diaphragm and adhesions of the basal pleura, it is difficult for pneumoperitoneum to achieve an adequate diaphragmatic elevation; however, since that is not always the case, such a condition is not a formal contraindication.

7) Pregnancy is not a contraindication, but rather a complication and each case must be worked out individually. When pregnancy occurs in the course of pneumoperitoneum, treatment should be gradually abandoned by reducing the amount of refills, since the growth of pregnant uterus replaces the amount of air needed to maintain an adequate diaphragmatic elevation. Before the uterus reaches the vicinity of the umbilicus, refills should be suspended and resumed as soon as possible after childbirth.

SUMMARY

Although there is no doubt that pneumoperitoneum is, in competent hands, a relatively safe procedure, well tolerated, free from serious complications and capable of favorably influencing the course of pulmonary tuberculosis in many patients, the final evaluation of a therapeutic procedure demands considerations not only of the immediate results but also of its ability to bring about a definitive arrest of the disease.

The advantages of pneumoperitoneum are evident in the treatment of bilateral lesion, with or without cavity, not too far advanced, where no other treatment can be performed because of the extent of the disease; in patients with acute, active and progressive tuberculosis who are too ill for immediate surgical treatment and in whom the risks of serious complications from pneumothorax are especially high; in patients with relatively small lesions where bed rest and modern chemotherapy are insufficient and in which pneumothorax could not be performed and the type of disease does not justify the employment of permanent collapse or pulmonary resection.

RESUMEN

Aunque el neumoperitoneo es sin duda un procedimiento seguro en manos competentes, y es además bien tolerado y está libre de complicaciones serias así como es capaz de influir favorablemente sobre la evolución de la tuberculosis pulmonar, la estimación final de su valor terapéutico requiere consideraciones no solo de los resultados inmediatos sino de su capacidad para conducir a la detención definitiva de la enfermedad.

Las ventajas del neumoperitoneo son evidentes en el tratamiento de las lesiones bilaterales con o sin cavidad, no demasiado avanzadas, en las que ningún otro tratamiento puede practicarse por lo extenso de la afección; en pacientes con tuberculosis aguda activa y progresiva que se encuentran demasiado enfermos para soportar de inmediato el tratamiento quirúrgico y en los que el riesgo de serias complicaciones del neumotorax es especialmente elevado; en enfermos con lesiones relativamente pequeñas en los que el reposo en cama y la quimioterapia moderna son insuficientes y en los que el neumotorax no se ha podido realizar y la forma de la enfermedad no justifica el empleo del colapso permanente o de la resección.

RESUME

Bien que, sans aucun doute, le pneumopéritoine, entre des mains compétentes, réalise un procédé relativement sûr, bien toléré, sans complications

sérieuses, et capable d'influencer de façon favorable l'évolution de la tuberculose pulmonaire chez beaucoup de malades, l'évaluation finale d'un procédé therapeutique demande que soient considérés, non seulement les résultats immédiats, mais aussi les possibilités d'amener l'arrêt définitif de la maladie.

Les avantages du pneumopéritoine sont évidents dans le traitement des lésions bilatérales, avec ou sans cavité, pas trop graves, mais où aucun autre traitement ne peut être utilisé à cause de l'extension des altérations; chez les malades qui sont atteints de tuberculose aigue active et progressive, dont l'état est trop grave pour un traitement chirurgical immédiat et chez lesquels il y à des risques particulièrement importants de complications graves si l'on entreprend un pneumothorax; chez les malades porteurs de lésions relativement peu étendues, pour lesquels le repos intégral au lit et la chimiothérapie actuelle restent sans effet, pour lesquels le pneumothorax ne pourrait pas être pratiqué, et dont le type de la maladie ne justifie pas l'utilisation d'une collapsothérapie permanente ou d'une exérèse pulmonaire.

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Report of a Case of Systemic Moniliasis*

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It is with no little concern that Medical Technologists have noted the more frequent occurrence of fungi in material submitted to the clinical laboratory for bacteriological study. Until recently not too much thought was given to these higher forms of bacteria, but today we view the situation with trepidation.

Within the last six months a rising incidence of mycotic infections, primary as well as secondary, has been observed. One of the common offenders has been the monilias, including the pathogenic strain Candida albicans and even those formerly listed[1] as non-pathogenic: Candida tropicalis, Candida Krusei, and Candida parakrusei. Their cultural characteristics have been typical in reaction and appearance but grew out in a remarkably shorter period of time. Characteristic growths were obtained in 36 to 48 hours on blood agar, Sabouraud's, brain heart infusion, Littman's, and thioglycolate. These monilias have been found not only in their usual habitat of mouth, throat, sputum, vagina, etc., in increasing numbers, but also in the urinary tract, and, in one instance, fulminating throughout the body. The report of this case follows:

Case 44-3383: This 54 year old white female was admitted to the hospital on January 3, 1952, with the complaint: coughing, spitting up blood, and broken out around the mouth. She gave a history of weight loss and anorexia of several months' duration following an episode of pleuritic pain in September, 1951. For the four weeks preceding admission she had had a blood-tinged, purulent sputum, night sweats, frequent spells of sore thorat, and pain in swallowing. As far as is known she was afebrile during this time. She had received unknown amounts of various broad spectrum antibiotics with no demonstrable response.

Past History: gall-bladder and appendix removed in 1944; mild asthma since childhood.

Family History: She had been living with a 35 year old brother who is ill and who has a severe cough.

Physical Examination: An acutely ill white female with numerous lesions of the skin and mucous membranes. These were most marked about the lips. The lesions were granulomatous elevated areas 1 to 1½ cm. in diameter. The tongue was red and the pharynx injected. No masses or enlarged lymph nodes were noted about the neck, but the trachea was deviated to the left. There was diminished expansion of the left thorax with slight impairment upon percussion. Fine rales were heard throughout the left chest. The spleen was not palpable. The remainder of the physical examination was not contributory.

Chest films (Figures 1 and 2) were made the morning after admission. Report is as follows:

"The cardiovascular shadows show no significant abnormality. There is a heavy wedge of infiltration in the left upper lobe posteriorly. Additional patches of density are noted in both bases, and small areas of increased density are noted in

*From St. Joseph Infirmary, Louisville, Kentucky.

the upper right lung. The trachea appears to be displaced toward the left. Slight generalized increase in density throughout the left lung is noted in comparison with the right. Some thickening of pleura at the left base is suggested with obliteration of the costophrenic sinus. The right hemidiaphragm shows no abnormality. The bony thorax appears normal.

"Conclusion: Pulmonary tuberculosis affecting chiefly the posterior segment of the left upper lobe with scattered basalar areas of infiltration and right upper

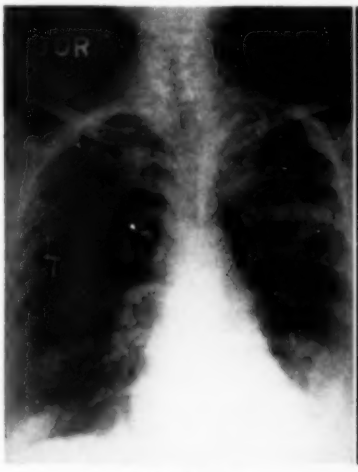

FIGURE 1

FIGURE 2

Figure 1: A-P view of the chest.
Figure 2: Lateral view of the chest.

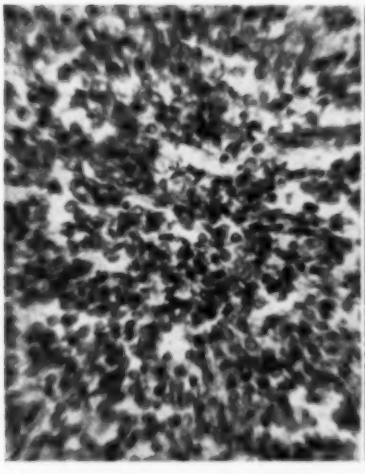

FIGURE 3

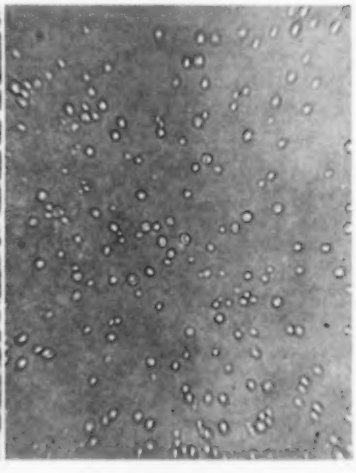

FIGURE 4

Figure 3: C. albicans—Gram's stain.
Figure 4: C. albicans—wet preparation.

lobe involvement is suggested. Several of the areas appear rather rounded in contour and metastatic neoplasm cannot be excluded. Other infectious processes such as fungus lesion should also be considered."

Laboratory Data: On admission blood count showed hemoglobin 10.7 grams, 3,800,000 erythrocytes, 9,000 leucocytes with 78 per cent polymorpholeucocytes, 19 per cent lymphocytes, and 3 per cent eosinophiles. Urine analysis: Dark, cloudy amber, pH 5.0, specific gravity 1.007, two plus albumin, sugar negative, and acetone negative; microscopic: few squamous epithelial cells, few red cells, few white cells, and uric acid crystals. Kahn report was negative. Sputum examinations submitted on January 4 and 5, 1952, showed presence of budding yeast bodies, mycelia, and many pus cells. No acid-fast bacilli were seen in the concentrated specimen. Culture produced growth of C. albicans in 24 hours (Figures 3 and 4).

Course in Hospital: Temperature on admission was recorded as 102.4 degrees F. orally. She pursued with febrile course throughout her hospital stay (Figure 5). Initially she was given a transfusion and general supporative therapy. Following the completion of the admission sputum examination, it was thought that a fungus infestation was the most probable cause of the patient's illness, and she was placed on a regime of 10 drops of saturated solution of potassium iodide, and other general measures were taken. She was seen in consultation by a dermatologist who states he believed that the cutaneous and mucus membrane lesions represented an infectious granuloma, and he performed biopsy of one of the areas on the lower lip. Subsequent study of the section of the biopsy confirmed this opinion. Within 24 hours after the initiation of the iodide therapy a marked progression of the patient's visible lesion took place, and her respiratory difficulties increased greatly. She shortly developed clinical signs of respiratory obstruction. Bronchoscopy was performed at this time, and similar granulomatous lesions were found involving the larynx, trachea, and major bronchi. It was believed that the iodides might have been the cause of this sudden progression of the disease, and they were discontinued less than 72 hours after the first dose. She was placed on Cortisone and received one dose of 100 mgms. Her obstructive symptomology continued to progress in spite of oxygen therapy, and she expired on her eighth hospital day.

Post-mortem examination revealed the major internal pathology confined to

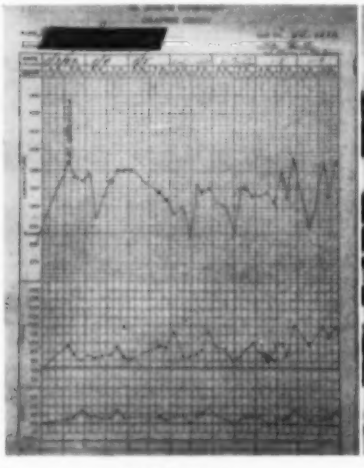

FIGURE 5

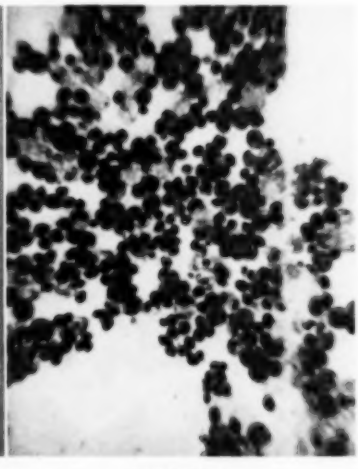

FIGURE 6

Figure 5: Graphic chart.
Figure 6: Section of spleen—H. and E. stain.

the respiratory tract, spleen, and kidneys. The middle and lower lobes of the right lung were completely consolidated. Cut sections showed a firm, grayish surface from which a sero-purulent material exuded profusely. Both lobes in the left were extensively involved in a similar process. The major bronchi were almost completely occluded by multiple granulomatous lesions. The spleen weighed 400 grams and presented no normal architecture. It was replaced completely by an inflammatory process identical with that in the lungs.

Microscopic examination: histologic sections showed fungi characteristic of monilia in granulomatous processes in the lungs, spleen, kidneys, and skin. The spleen was particularly interesting in that the fungi appeared to be growing in large colonies (Figure 6).

The pathologist's conclusion was generalized moniliasis primary in the respiratory tract.

SUMMARY

A case of Systemic Moniliasis has been reported outlining laboratory and clinical data, treatment, hospital course, and subsequent autopsy findings. What part previous antibiotic therapy may have played in this case is left an open question.

RESUMEN

Se ha relatado un caso de moniliasis generalizada subrayando los datos clinicos de laboratorio, la evolución en el hospital y la autopsia consecutiva con sus hallazgos. Queda abierta la cuestion del papel que la terapéutica previa con antibióticos ha desempeñado en este caso.

RESUME

Les auteurs rapportent une observation de moniliase et en définissent les éléments cliniques et biologiques, le traitement, l'évolution à l'hôpital et les constatations d'autopsie. Ils se demandent quelle part à pu jouer dans ce cas un traitement antérieur par les antibiotiques.

REFERENCE

1 Martin, D. S., Jones, C. P., Yao, K. F. and Lee, L. E. Jr.: "A Practical Classification of the Monilias," *Jour. Bact.*, 34:99, 1937. As quoted by Dubos, Rene J.: "Bacterial and Mycotic Infections of Man," J. B. Lippincott Co., Phila., 1948, p. 602.

An Inexpensive Homemade Balloon Cuff for Endotracheal Tubes

DAVID J. SOBIN, M.D., M.S.*
Los Angeles, California

In 1893, Maydl[1] conceived of a plan for introducing an anesthetic through a tracheal tube; and Eisenmenger[1] added an inflatable cuff around the distal end of the tube. However nothing was done with these ideas for over a quarter of a century. In 1924, Waters[2] described the "carbon-dioxide absorption" method of anesthesia, and four years later Waters and Guedel[3] published an article on the "Intratracheal Catheter" in which they described a cuff to be made either by cementing a piece of rubber sheeting around the catheter or by a double layer of rubber slipped tightly over the tube.

We have constructed a rugged cuff with material found in and around the hospital at the cost of only a few pennies.

Materials used:

1) A piece of doweling one-half inch in diameter.
2) One-half inch Penrose tubing at least four inches long.
3) A catheter size No. 10.
4) Rubber cement (See Figures 1 to 5).

Method:

1) Make a notch in the dowel one and one-half inches long and deep enough to accommodate catheter.

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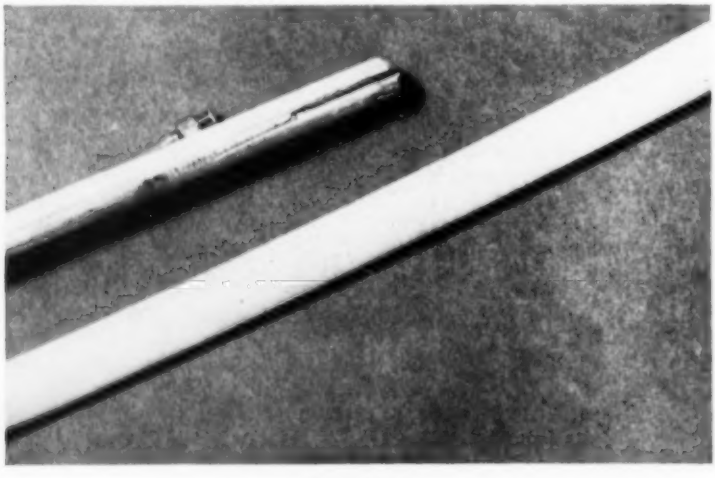

FIGURE 1

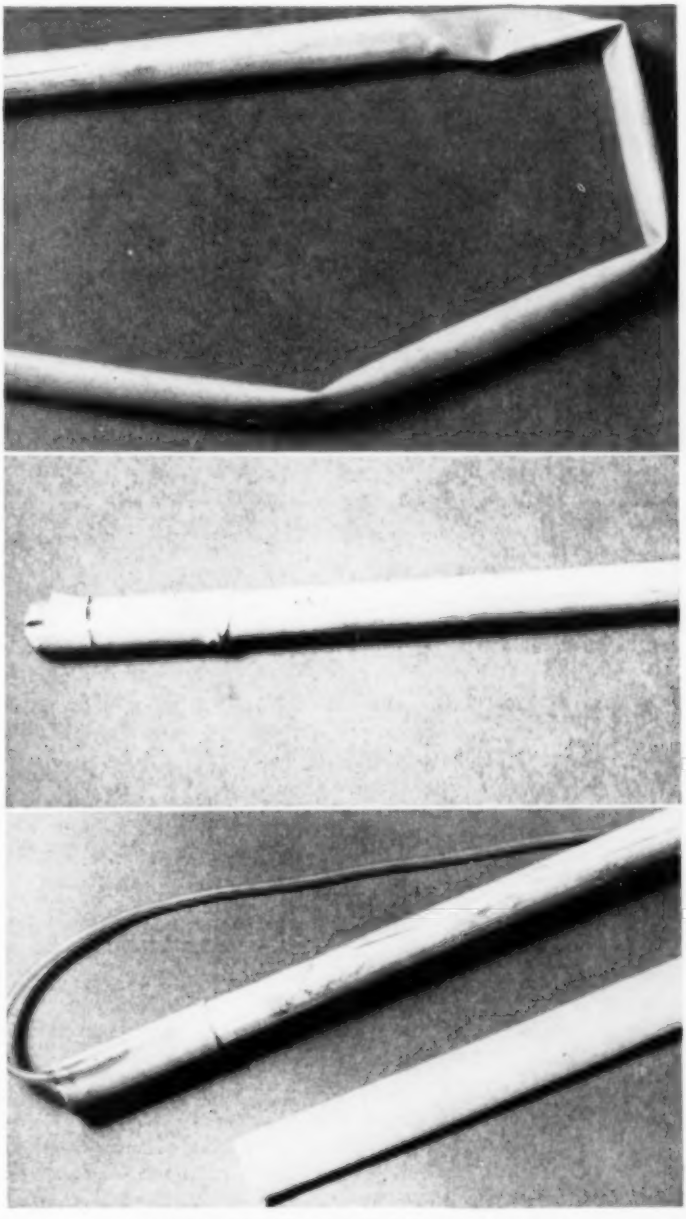

FIGURE 2 (top).—FIGURE 3 (middle): The black line is merely a demarcation of the turned back edge with ink).—FIGURE 4 (bottom).

2) Slip the penrose tubing over the notched end, and two inches past the notch.

3) Fold the Penrose tubing back on itself.

4) Insert the catheter between the layers of the Penrose tubing, to the end of the notch.

5) Apply cement between layers of Penrose tubing and around catheter for a distance of one inch. Roll on flat top or table to spread. Let stand overnight to dry. Slip tubing and catheter off dowel, and balloon cuff is ready for use.

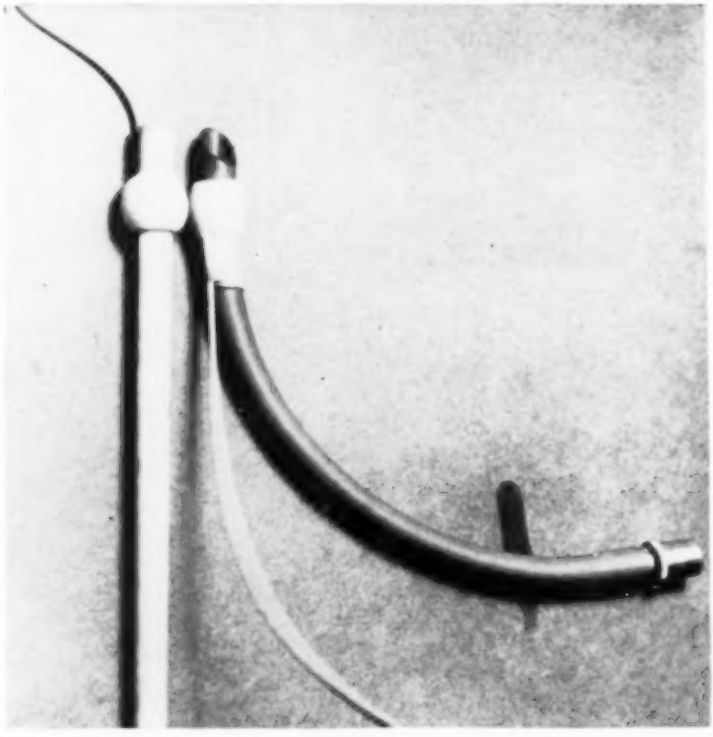

FIGURE 5

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1 Robinson, Victor: "Victory Over Pain," New York, 1946, p. 282.
2 Waters, R. M.: "Current Res." *Anesth. and Analg.*, 3:20, 1924.
3 Waters, R. M. and Guedel, A. E.: "Current Res." *Anesth. and Analg.*, 7:238, 1928.

Editorial

SOME SERIOUS CONSIDERATIONS ON THE TREATMENT OF TUBERCULOSIS

No more important article on the treatment of tuberculosis was published in 1952 than that which appeared in the July issue of the *World Medical Association Bulletin* by Kjeld Torning, Chief of the Oresundshospital of Copenhagen, and President of the Danish Society of Phthisiologists. This paper should have a sobering influence on any tendency to make sweeping statements concerning therapeutic procedures without evidence based on well controlled groups of patients over sufficiently long periods of observation. The medical profession should not assume credit for certain favorable changes in the tuberculosis situation unless there is evidence that it has been at least partially responsible. For example, Dr. Torning directs attention to the mortality rate from tuberculosis in Copenhagen from 1870 to 1900, which decreased from 300 per 100,000 to 150, yet there was no organized prophylaxis or was there any effective treatment during this period. Similar changes occurred in other parts of the world, without much participation of the medical profession.

He emphasizes that in a capricious disease like tuberculosis, the only way to get a reliable quantitative expression of the value of some form of treatment is to earmark a large group of persons as uniform as possible for experiment and give every other one the treatment in question, taking care that the two groups apart from this are treated alike, and later the results are compared. Unfortunately, in the past we neglected to apply such a method to large categories of patients treated in various ways. Therefore, today, we have almost no accurate, well documented information on any of the forms of treatment. Nevertheless, we express opinions freely about treatment methods of the past and the present. All too often, just opinions of persons with worldwide reputation have been accepted as established facts and in notable instances the medical profession has been misled.

In presenting the evolution of therapeutic pulmonary collapse, Torning states that artificial pneumothorax is still the dominating type of collapse treatment. He says: "If one calls any air anywhere in the pleural sac a therapeutic pneumothorax, one is asking for trouble and if one gets it, not the treatment but the physician is to blame." He points out that in properly selected early cases one may expect even without drugs a lasting effect in some 80 per cent in those cases where satisfactory pneumothorax can be established. "No other method conservative or surgical has been proved to yield more." He reminds us that pleural complications which sometimes result in impairment of pulmonary function are strongly influenced by present day drugs which presumably in other respects also will improve late results considerably and thus increase the desired results to well over 80 per cent of the persons so treated. In this issue of *Diseases of the Chest*, Dr. D. G. Birath, Gothenburg, Sweden, reports marked reduction in the com-

plications of artificial pneumothorax when preceded and accompanied by drug administration. There are many thousand people living and well in the world today who overcame tuberculosis by the aid of artificial pneumothorax. In all probability, this will remain an excellent form of treatment in many parts of the world for a long time. Therefore, it is exceedingly important that Birath has shown that most of its major complications can be prevented.

Torning points out that the efficacy of thoracoplasty depends to a large extent on indications, "If everybody who has a fair chance to benefit from thoracoplasty is operated we may according to the experience in our hospital (Hagn-Meincke) count on curing some 50 to 60 per cent of cases which, without surgical treatment, would have only some 10 to 20 per cent chance of surviving." There is evidence that chemotherapy as an adjunct may definitely improve this result.

Concerning segmental pulmonary resection, he states that it is still premature to speak about established indications. It is impossible finally to judge the value of any treatment in pulmonary tuberculosis until after several years of observation. Moreover, one cannot compare results of resections done under the protection of drugs with those of pulmonary collapse before drug treatment was introduced. In a few places artificial pneumothorax has been abandoned in favor of pulmonary resection. The wisdom of this change from one method to the other cannot be known until one-half of a large group of similar cases is treated by one method and one-half by the other while all other factors, including chemotherapy, are alike for the entire group. Such a study would need to be continued 10 years and preferably much longer

Dr. Torning points out that of all the preparations recently introduced, streptomycin and para-aminosalicylic acid in combination are the best. Whether these drugs can cure the disease or merely prolong life is still uncertain. "The steep drop in mortality in Copenhagen in the last two years no doubt is due to a considerable extent to the fact that drug treatment has postponed but not averted a fatal issue in many desperate cases." Moreover, in some cases in which surgical procedures were out of the question a few years ago, improvement and even restoration to good health is now possible under cover of drug treatment.

In the past, enthusiasm reigned supreme for certain forms of treatment such as fresh air, climate, altitude, over-feeding, strict bed rest for months and years, tuberculin and other drugs. Proponents of these and many other methods made sweeping statements pertaining to efficacy without factual data. Although some of the greatest minds in medicine have been deceived by the tubercle bacillus and their overenthusiasm for therapeutic procedures later proved unjustified, the temptation still exists, and at this moment great names are being risked by expressions of opinions about therapeutic methods based on inadequate evidence. Torning's emphasis on the time element is apropos. With a lifetime vicissitudinous disease noted for remissions and exacerbations it is impossible to accurately determine the efficacy of any form of treatment in a few years. Indeed, a decade is a short period.

Torning says, "We must, therefore, confess that in spite of the unlimited material of patients available we can produce few figures to prove the efficacy of different treatments. Far too often we have to confine ourselves to state what we feel or believe." This authentic statement is a serious reflection on our past and present deportment.

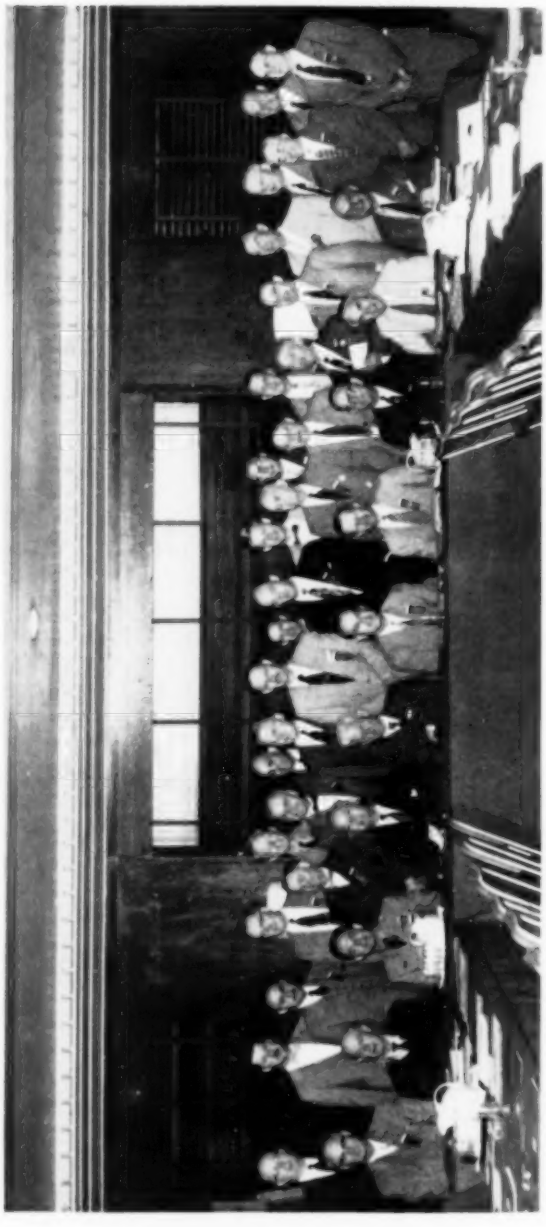
A good start has been made—probably the best in history—to conduct well controlled studies in chemotherapy. Some of them meet Torning's criterion regarding comparability of cases. None has been carried on long enough to justify more than tentative conclusions, but continued observations of the same cases should ultimately result in important facts. Tuberculosis is one of the diseases in which almost painful patience must be exercised by the physician in determining efficacy of any therapeutic measure. To draw conclusions without making observations on a large material over at least a decade is likely to be hazardous.

Those who believe that despite marked decreases in mortality there has been increase in morbidity will be interested in the Copenhagen figures and Dr. Torning's explanation. In 1920 the mortality rate was 93, but in 1950 only 24, approximately 25 per cent of what it was 30 years ago. However, the number of new cases reported each year is only slightly altered. This does not mean that morbidity has been unaltered. "Thanks to the growing intensity of case finding a much larger percentage of existing cases are known today than 30 years ago—and a much larger proportion of today's cases are accessible to treatment "

One of the most significant phases of Torning's article is his recognition of the paramount importance of the general practitioner in the tuberculosis control program. With reference to touring teams conducting certain procedures, he says, "It would be a catastrophe if such measures created in the minds of general practitioners the feeling that tuberculosis is something which others take care of. The practitioner deals with sick people and tuberculosis is more common among people who feel ill than among those who feel well. He is the man who first sees the sick person and who knows his environment. Therefore, the tuberculosis-minded general practitioner is the cornerstone in the fight against tuberculosis."

J. Arthur Myers.

ANNUAL MEETING, BOARD OF GOVERNORS



Governors of the College meeting in annual session, New York City, May 28, 1953.

Annual Meeting, Board of Governors

The Board of Governors of the American College of Chest Physicians met in annual session at the Hotel New Yorker, New York City, on Thursday, May 28, at the time of the 19th Annual Meeting of the College. Dr. William R. Rumel, Chairman of the Board of Governors presided. Dr. Edward W. Hayes, Chairman of the Council on Undergraduate Medical Education of the College addressed the Board on "The Teaching of Diseases of the Chest in our Medical Schools." Dr. David H. Waterman gave a brief talk on "The Value of Visual Aids in Teaching Diseases of the Chest." A report on the College Membership was presented by Dr. Harold G. Trimble.

Dr. William S. Conklin, Portland, Oregon was elected by the Board of Governors to membership on the Committee on Nominations for the year 1953-1954. Dr. David H. Waterman, Knoxville, Tennessee was unanimously elected Chairman of the Board of Governors for the coming year.

Award of the 1953 College Medal to Dr. Helen Taussig

HAROLD GUYON TRIMBLE, M.D., F.C.C.P.
Oakland, California

I am especially privileged this evening. First, for the opportunity of speaking for the College in presenting this annual award. Second, for the real thrill that was mine in reviewing the material for this presentation.

The previous recipients of this medal form a distinguished group: our own Dr. J. Arthur Myers, for his leadership in the epidemiology of tuberculosis and many other contributions; Dr. Charles M. Hendricks for his distinguished services to the College; Dr. Evarts Graham, the distinguished surgeon; Dr. Manuel de Abreu, Brazil, an outstanding scientist who is truly responsible for initiating the mass x-ray survey, and more recently for developing bronchial lavage as a diagnostic method; Dr. Selman Waksman for his discovery of streptomycin; Sir Alexander Fleming for his penicillin; and Dr. Jorgen Lehmann for opening up a whole new vista of tuberculosis therapy with carefully planned research in chemotherapy, and the development of para-aminosalicylic acid compounds; and Dr. Chevalier Jackson, for meritorious contributions to diseases of the chest.

Your Committee on College Award composed of Dr. Benjamin L. Brock, Orlando, Florida, Dr. R. O. Joplin, Louisville, Kentucky, and myself as Chairman, appointed by your Regents a year ago, had but little difficulty in reaching unanimous agreement. It was necessary that the recipient know of this award in advance. You all know the unique merit of the work which we in a small measure wish to again commend by this award. We felt there must be some story behind this. How did this come about? It could be no accident. In searching about for such information, I made no real headway; so I decided to go directly to the recipient, and that was done. In due course I received a reply to my questions and I was thrilled as this story unfolded before me. I hope that I can impart to you the excitement that I felt at that time.

I must first of all tell you that the recipient of the Medal tonight is a woman.

"I am the daughter of a Professor of Economics of Harvard University, and had all of the advantages of growing up in a University atmosphere. After graduation from College, when I first seriously considered studying medicine, my father felt

the field of Public Health was a better field for women. Therefore he urged me to inquire into the then new Harvard School of Public Health, concerning their attitude to women. Dr. Rosenau, the Dean of the new school of Public Health, told me that the requirements for admission were two years in a recognized medical school; thereafter they would 'permit women to study but would not admit them as candidates for degrees.' I asked him who he thought would be fool enough to spend two years studying medicine and two more years in the school of Public Health and not get a degree. His reply was, 'No one, I hope.' My reply was 'I certainly will not be the person to disappoint you.' That really settled my going into medicine and my father was entirely content. He lived to be happy that I had done so.

I started my studies at Harvard University by taking histology under Dr. Bremer and then traveled in Europe for eight months with my father and sister. My father urged me to take a year abroad because as he said, once in medicine it would be a long time before it would be possible to travel again.

Dr. Bremer urged me to go to Boston University and take anatomy. So the following fall I entered the Boston University School of Medicine for this purpose. Just before Thanksgiving, Dr. Alexander Begg, who was Professor of Anatomy and Dean of the University, gave me an article by Franklin P. Mall on the Anatomy of the Muscle Bundles of the Heart, and told me to study that problem and added, 'It won't do any harm to be interested in one of the major organs of the body as you go through medical school.' I took the article home over the Thanksgiving weekend and read it on the train returning from the holiday. It was my first introduction to tough reading and I well remember returning to medical school with fear and trepidation for I understood very little of that

Presentation of the College Medal to Dr. Helen Taussig at the President's Banquet, Hotel New Yorker, Saturday, May 30, 1953. Standing left to right: Dr. Richard R. Trail, London, England; Dr. Taussig; Dr. Harold G. Trimble, Oakland, California, Chairman of the Committee on College Awards; and Dr. Alvis E. Greer, Houston, Texas, incoming President of the College.

article. Dr. Begg gave me a beef heart and told me to go to work. I read and reread the pages of that article, looked at the beef heart and explored a little here and a little there. Dr. Begg looked in occasionally, said nothing and walked out. Finally after a couple of days I had completely mutilated the heart and felt entirely discouraged. Dr. Begg came in and said, 'Well, you got further than most, we'll get you another heart.' Then I really settled down to work on the anatomy of the muscle bundles of the heart.

I studied anatomy until I was lost and then in innocence thought it might be easier to study physiology and started my work on isolated strips of cardiac muscle and succeeded in getting them to beat in oxygenated Ringer's solution.

The following year I spent partly in research and partly taking courses in medical school. I took physiology at Boston University, and then took bacteriology under Dr. Zinser at Harvard. It was Dr. Begg who urged me to go to Hopkins. So in the fall of 1924 I entered Hopkins in the second year with some advanced standing. My free time during the three years of medical school was spent in the heart station working with Dr. E. P. Carter. I applied for a medical internship and was turned down, and thereupon Dr. Carter offered me a fellowship to work in his laboratory for a year. Dr. Park came to Baltimore in the fall of 1927. He had a great interest in special clinics because he felt that was the way to study disease. He started the tuberculosis clinic, the psychiatric clinic and then the cardiac clinic. In starting this he consulted with Dr. Carter. In the spring of 1928 Dr. Benjamin Harris and I (both fellows in the heart station) worked in the Harriet Lane cardiac clinic. That fall (1928) I started my internship in Harriet Lane. The following spring Dr. Park asked me to take charge of the cardiac clinic. It was a young clinic and I was very young and I was really happy for the chance to come back to Baltimore. So in the fall of 1930, I took charge of this cardiac clinic, and was given a list of 200 names, a technician, and a social worker. We went to work and during that fall we got an electrocardiograph and later in the winter a fluoroscope was installed. It really was the first fluoroscope in the hospital that the house staff and the interns were allowed to use. Well I remember the trials and tribulations we had that first year. Nevertheless, Dr. Park continuously urged me to use it.

Dr. Park gave me the opportunity to learn pediatric cardiology. During that first winter he once remarked to me, 'Now, Doctor, you have got to learn congenital malformations of the heart.' I replied to him in some rather uncomplimentary terms to the effect of 'Dammed, if I will.' He said to me, 'It doesn't matter how you feel, it's going to be forced upon you.' Dr. Park insisted that I see every baby with any sort of cardiac difficulty and that we should get an electrocardiogram on each, and that I fluoroscope all my patients.

It was not long before I fluoroscoped a cyanotic infant in whom we could see no right ventricle and no projection anteriorly in the left anterior oblique. To my delight the electrocardiogram showed a left axis deviation; so I postulated that the baby had an absent right ventricle. That really created excitement and they followed that baby very closely. At autopsy the baby had no right ventricle! That was the beginning of my interest in congenital malformations of the heart.

As so often happens, things come by twos and threes. A couple of weeks later another baby was admitted. The x-ray was so much like the first that when it was put up, the resident said, 'Oh, excuse me, that is the wrong x-ray' and I replied, 'No, that is the right x-ray, but it is a different child with the same congenital malformations.' From then on every one in Harriet Lane got interested in congenital malformations. They got interested in trying to see whether or not I could diagnose them correctly. My effort in diagnosis got the house staff interested in trying to get autopsies in order to check them. It became a game and at x-ray conference we used to show the x-rays and discuss the findings and then, if we could, check them in pathology.

It was by studying the children and by watching them for a period of years

I began to realize that some of the cyanotic children died from lack of oxygen and not from cardiac failure. Then I gradually realized that the closure of the ductus in many a child with pulmonary atresia was the cause of death. Therefore, I realized that if one could put in a ductus it would help the child.

Naturally when Dr. Robert Gross showed that it was possible to ligate the ductus successfully, it gave a tremendous stimulus to all of us and I immediately realized that if you could put in a ductus it would help a number of cyanotic children. I was far more worried by the operation on the first child with a patent ductus arteriosus than about the first patient with a tetralogy of Fallot, mainly because I did not share Dr. Gross' belief in the poor prognosis of a patent ductus.

After Dr. Blalock's appointment as professor of Surgery and knowing that he was interested in thoracic surgery, it seemed wise and fair not to launch on any cardiac surgery until he came to Baltimore. In 1942 Dr. Blalock ligated the first ductus at Johns Hopkins Hospital and the third he had ever done. I well remember that experience and how much we both were on the spot for the diagnosis and for the surgery. How strange I felt when I looked into the chest and saw the heart beating and how difficult it was for me to get oriented to positions of the various vessels and to know what was what.

At the end of the operation I said to Dr. Blalock, 'I stand in awe and admiration of your surgical skill, but the really great day will come when you put in a ductus to increase blood to the lungs for the child dying of anoxemia and not ligate a vessel when there is too much blood going to the lungs.' Dr. Blalock sighed, he really thought he had done something that day. Then he said to me, 'When that day comes, this will be like childs play.' That was our start in surgery together.

Dr. Park had interested Dr. Blalock in the problem of coarction of the aorta and so I tried to interest him in cyanotic children and showed him various children who needed surgical help. He accepted the challenge and took it to the laboratory and he spent many, many days and many, many experiments trying to create pulmonary stenosis and doing vascular anastomosis and finally succeeded.

The rest, I believe, is known to you."

Well, indeed it is known to us. It is known not only to us but around the world. For fourteen years as physician in chief of the Harriet Lane heart clinic, this physician has been engaged in the study of youngsters' hearts. She liked doing it without intrusion in her little office off the clinic corridor, known for its "orderly disorder."

You know by now of course, that I'm speaking of Dr. Helen Taussig.

From this point on doctors came to Baltimore from all parts of the world to learn these methods. Blue babies were flown in from every place overnight. The world of medicine began to pay homage, and that part of it concerned with the hearts of children began to beat a path to this cluttered little office. The office, itself, looked like a clinical exhibit of the heart. Plastic replicas of hearts sit on the floor, on the desk, everywhere, and on the walls hang many pictures. The furniture appeared to be survivors of the first years of Hopkins. The first operation took place in November 1944. Since then the operations numbered in the hundreds. Dr. Taussig has examined children for this purpose numbering into the thousands.

It was not long until she was appointed Associate Professor of Pediatrics at Johns Hopkins. She was a member of the editorial board of the Journal of the American Heart Association. Much of this work had to be published. Then came the book on Congenital Malformations of the Heart, published by the Commonwealth Fund. It became the bible of physicians interested in this group of diseases. Numerous awards followed and a whole series of honorary degrees too numerous to detail at this time.

Summing all this up, it again illustrates a medical truism that significant medical advances are made when alert, well trained physicians acquire basic information through many years of hard toil. They take this knowledge, combine

it with advances in related fields, and with the cooperation of like minded colleagues, achieve significant advances in the relief of another human ailment.

Dr. Helen Taussig, the American College of Chest Physicians here tonight is proud to honor you by conferring upon you at this time its annual Medal for your pioneering achievements in the field of congenital heart disease.

Presentation of Honorary Fellowship Certificates

ANDREW L. BANYAI, M.D., F.C.C.P.

In awarding Honorary Fellowship Certificates to the men in charge of the Medical Services in various branches of our Armed Forces and in the Veterans Administration, we want to pay homage to the unparalleled splendid developments of preventive, clinical and field practice of medicine and surgery in these groups.

Because we recognize that the present day excellent standing of these Medical Services is the result of the brilliant work of the men in charge of these enormous units, as a token of our admiration, we are bestowing upon them Honorary Fellowship in the College.

Also, we consider this occasion a fitting opportunity for honoring one of the greatest living clinicians of America.

May I say on behalf of the officers and of the entire membership of the College that by honoring these distinguished gentlemen we are actually honoring the College and honoring ourselves.

PAUL D. WHITE, M.D.
Executive Director, National Heart Advisory Council
and Chief Consultant, National Heart Institute.
Boston, Massachusetts

It is with great appreciation on the receipt of this honor that I welcome the opportunity to present a few thoughts that have been on my mind for a long time concerning specialization and special medical clinics and societies. These ideas, based in large part on my own experience, may be, I hope, of interest and help to others. They are presented without egoism or egotism although the relation of this factual material may at times seem to be such.

I would like to go back four decades. The first, 1910 to 1920, was the period when special medical research laboratories in hospitals and schools in this country began widely to be set up. This was the first light of the new dawn in a hospital like the Massachusetts General. I happened to be personally one of the first actors in that first scene, having been given the position of the second medical resident. The first medical resident of all there at the M. G. H. was Walter Palmer, later Professor of Medicine at Columbia. Our duties were to develop medical research, not to supervise the internes, although we were on call to advise them. Meanwhile, James Howard Means, although not a resident, was setting up his own thyroid laboratory. We all had very small, out of the way quarters for our first years. My own was a closet in the basement of the old skin Ward G next door to the bathroom reserved for syphilitic patients. These were, of course, all trial balloons, many of the staff not believing that anything really would ever come of it, especially of my own use of that very doubtful medical toy, the electrocardiograph. A sign of the times was that I was designated to give a series of lectures on what was then known about electrocardiography to my medical chiefs and professors at the Harvard Medical School.

AWARDING OF HONORARY FELLOWSHIPS

Convocation, 19th Annual Meeting, American College of Chest Physicians, Hotel New Yorker, New York City, Saturday, May 30, 1953.

Pictured, left to right: Dr. Paul D. White, Boston; Major General George E. Armstrong, MC, USA, Washington, D. C.; and Dr. Leonard A. Scheele, Washington, D. C. Dr. Andrew L. Banyai, Milwaukee, President of the College, made the presentations.

The next decade from 1920 to 1930 was even harder because now some of us had the temerity to establish, almost surreptitiously, special clinics and special practices frowned upon for the most part by our chiefs and professors and indeed by many of our colleagues who wanted internal medicine and surgery to remain "general." I had to fight this battle for years but the restraint that was put upon me had one very good result. We were obliged to have a share in the general medical wards and Out-Patient Department clinics and constantly to rub shoulders with our colleagues in other fields. This compromise was, I am sure, wise and I am glad today that I did not try to break away to establish a Cardiac Institute of my own, in my opinion an undesirable goal despite the excellence of such a place as the National Institute of Cardiology of Mexico.

It was during that decade also that some of us had the temerity to establish the American Heart Association, an action adversely criticised by several of my very worthy contemporaries. For several years I carried on a most interesting debate by correspondence with three or four prominent professors here and there in the country who were strongly opposed to my special activities but who during the last ten years have fallen into line and have done much themselves for cardiology. You would be much interested and surprised, I know, if you should hear their names.

Also, early during that decade a group of my cardiological colleagues, friends, and students began to assemble with me regularly in my laboratory every week or two to discuss our problems in research and in the clinic. Among them were Sam Levine, Howard Sprague, Burt Hamilton, Soma Weiss, Cob Palmer, Larry Ellis, and Herrman Blumgart. From this beginning there developed the New England Heart Association but for many years after that (until the Second World War prevented our continuing this custom) we continued to meet as a "small heart group" in each others houses once a month. I am sure that a potent reason for the very friendly cooperative cardiological atmosphere that exists today in Boston was this long perpetuated custom.

The next decade, 1930 to 1940, saw a great expansion of the specialties, in clinic, practice, and teaching and their infinite ramification into subspecialties and subsocieties. All this was of the greatest importance. Cardiology (even the term) gradually became respectable and popular. The golden age was beginning to blossom.

And now during the last decade or more (1940-1953) there has come the almost full flowering of the specialties, both medical and surgical. The American Heart Association has had to develop several different councils and sections, rheumatic, hypertensive, and arteriosclerotic, and I have had to divide up my multiple responsibilities among many colleagues, for example, in electrocardiography, cardiac catheterization, the rheumatic problem, hypertension, etc. I feel like a very general practitioner now in the field of heart disease.

However, the multiplicity of medical drives, medical hearings, and special interests, confuses the public, the Congress, the Bureau of the Budget, and even the doctors themselves. Everywhere we are begged to begin "to put the body back together again." We must take more and more cognizance of these pleas, but meanwhile we must recognize that there is still a place and an important one, for both the detailed specialties and for general medicine and for all or nearly all the societies represented thereby. We must, of course, pick and choose ourselves according to our interests, our duties, and our available time.

The third category of societies and meetings which is the newest group of all is represented by this very College of Chest Physicians which is combining closely related organs and systems and "basic" scientists, internists, pediatricians, roentgenologists, and surgeons. I am delighted to be asked to be a Fellow of this newest type of Medical Society. I speak of it as representative of the newest type even though it is now 19 years old.

Finally, I would like to say a word about the National Heart Institute and the

National Advisory Heart Council of the U. S. Public Health Service with which I have been in part time associated during the past four years. It is a pleasure to see here this evening among my colleagues representing their national medical services, Surgeon General Leonard Scheele of the Public Health Service. In close cooperation with the American Heart Association, which is the greatest private cardiological agency in the world, and with the International Society of Cardiology, these bodies have a wide interest embracing all the groups which I have mentioned. It is a great pleasure to acknowledge their usefulness.

Again let me thank you very much for this honor which you have given me tonight.

GEORGE E. ARMSTRONG, Major General (MC) USA
The Surgeon General, United States Army.

I feel signally honored by being tendered a Fellowship in the American College of Chest Physicians and accept same, not for myself alone, but for the entire Army Medical Service. During the past few years we have seen a marked improvement in the standards of medical care within the Military Service. This, of course, has been in consonance with similar progress throughout the field of American medicine.

I am quite familiar with the aims and ideals of the American College of Chest Physicians and am cognizant of the part which this organization has played in this general improvement of medical care. We in the Military have felt the favorable impact of your contributions and wish, in turn, to extend to you our fullest support.

H. LAMONT PUGH, Rear Admiral (MC) USN
The Surgeon General, United States Navy.

It is with great pride that I accept this certificate of Honorary Fellowship in the American College of Chest Physicians and the responsibility the act of acceptance implies. I feel certain that this honor reflects appreciation, on the part of the officers of the College, for the devotion to duty of the members of the Medical Department of the Navy in the prevention, control and treatment of chest diseases and in the research efforts they have made in furthering medical knowledge in these fields.

On behalf of these earnest workers, many of whom are enrolled in the membership of this distinguished body, it is my privilege to express deep gratitude for the advice and support that Fellows of the College have unstintingly provided in the development of sound programs and policies. With their continued interest and support, which we shall strive to deserve, the future prospect is indeed bright that tuberculosis, that historically notorious scourge of seafarers, will continue to be an ever-diminishing threat to the health and security of the Navy.

Again I wish to express my appreciation for the great honor bestowed upon me.

HARRY S. ARMSTRONG, Major General (MC) USAF
The Surgeon General, United States Air Force.

I accept with great pleasure Honorary Fellowship in the American College of Chest Physicians. Since its inception in the middle 30's, this organization has provided leadership and a meeting place for all workers interested in the growing problems of respiratory diseases. As one reviews the status of chest diseases and respiratory problems of that time, the need for the College can be appreciated. Special attention to anesthesiology had developed knowledge and techniques in this area to the point where surgical therapy could be successfully applied in dealing with diseases of the chest. Hospitalization for the then major chest disease

—tuberculosis—was at its peak. Industrial problems dealing with dusty occupations, sand blasting, rock drilling, and the like, were becoming acute. Technical advances had permitted expansion of under water construction activity. The importance of submarine warfare in our national defense had been constantly growing. Aviation, both civil and military, had passed the stage where visual and equilibrium problems ceased to be as acute as respiratory problems in operation at high altitudes. Physiology and chemistry had developed relatively rapid simple techniques for research in the respiratory field.

Formation of the College focussed attention to the need for increased training in the area of respiratory physiology and successfully worked toward raising the standards and enthusiasm of physicians working in this area. The Nation, through its Armed Forces Medical Services, profited considerably as a result of these efforts in World War II. During this war, the ability to deal with wounds and diseases of the chest was greatly enhanced. The impetus the war gave to our ability to deal with diseases of the chest now includes considerable success in dealing with chest malignancies and more recently cardiac surgery. This rapid growth demonstrates the usefulness of an organization which brings together all of the specialties—medicine, surgery, anesthesiology, endoscopy—and the basic sciences. The American College of Chest Physicians was the first, I believe, to unify these specialized endeavors into one specialty group.

The government services have been important contributors to the rapid development of our ability to better diagnose and treat diseases of the chest. The Army and the Veterans Administration medical services' contributions to advances in the management of tuberculosis and fungus diseases of the chest are well known to all those present. The Public Health Service has made most important contributions in the control of the pneumoconioses and the prevention of industrial fume and dust hazards. The Navy and the Air Force medical services have been major contributors in the rapidly developing field of aviation medicine which advances both the military and civil aspects of flying. I cite the roll of our governmental services in advancing the work of your College so that the unity of endeavors become all the more apparent and I deeply appreciate the sentiment which motivated the College of Chest Physicians to bestow this honor on all of the titular heads of the Government medical services at this convocation. My personal gratitude is great.

LEONARD A. SCHEELE, M.D.
The Surgeon General, United States Public Health Service.

I am proud to have been selected as an Honorary Fellow of the College and to be associated in this way with your organization.

To my mind, the American College of Chest Physicians truly exemplifies the high ideals of a professional society to promote the science and perfect the art of its specialty. These ideals are realized in your constant endeavor to push forward the frontiers of medical knowledge, in your dissemination of that knowledge through medical education, and your vigorous efforts to improve the standards of medical care.

Since its founding, the membership of the College has steadily widened, as the narrow boundaries of disciplinary specialization have given way to a broader and more fertile outlook. And as the College has increased in strength, the variety of worthy projects carried out in its name have caused it to grow in esteem and stature.

I have in mind, for example, your continuing program to improve the teaching of chest diseases in our medical schools. The materials prepared for undergraduate instruction and the ready assistance offered to medical faculties help to discharge, in an outstanding way, your obligation to the practitioners of the future.

I am aware, too, that postgraduate education has also been prominent in your activities. The many papers and talks delivered under your auspices by leading chest specialists serve to keep your colleagues in general practice informed. Within the specialty itself, postgraduate courses and your invaluable journal, *Diseases of the Chest*, achieve the same indispensable end.

The accomplishments of the College in its program to raise the level of sanatorium construction and operation are well known. The medical management of patients with chest disease has also been improved, through the careful collection and evaluation of information about the effects of the antibiotics and the newer chemotherapeutic agents. Today the specialty practice of diseases of the chest is universally recognized.

As a public health physician, I have been especially interested in your strong efforts to further the control of tuberculosis through public health measures. Your proposal in 1940 that the Selective Service System x-ray all inductees, and your cooperation in carrying on this greatest of screening surveys, was a contribution of inestimable value to the Nation's health. The exchange of ideas with the American College of Radiology, which resulted in the formulation of principle to guide mass chest x-ray programs and hospital admissions, also contributed to public health progress. Your present campaign to strengthen tuberculosis control programs in penal and mental institutions is attacking a problem which has long been of deep concern.

I have observed too, that through the years the College also has been alert to the international responsibilities borne by a scientific society. The foreign chapters of the College, the bilingual summaries published in your journal, the opportunity to meet foreign colleagues at your international Congresses—all demonstrate your firm conviction that disease respects no boundaries and medicine acknowledges no barriers to the relief of suffering.

As I reviewed the growth and progress of the College in the relatively few years since its founding, I was mightily impressed by the number of points of contact with the Public Health Service. In many ways, the activities of the College have served to inspire, to stimulate and to assist the Service in the conduct of its duties. For this reason, I should like to accept the honor which you have bestowed upon me, in the name of the Public Health Service, since surely I was chosen by virtue of my position as its representative. On behalf of the Public Health Service, then, I accept with gratitude and pride this Honorary Fellowship in the American College of Chest Physicians.

JOEL T. BOONE, Vice Admiral (MC) USN, Retired
Chief Medical Director, United States Veterans Administration

From the very first annual meeting of the American College of Chest Physicians, career physicians of the Veterans Administration have actively participated in the scientific deliberations of the College, and for the last dozen years the Veterans Administration, with pride, has been officially represented on your Board of Governors. In the interim since its incorporation we have seen the College develop, first nationally, as a potent force for advancement of learning in pulmonary diseases, and then, internationally, as a good will ambassador drawing together the physicians of many countries in a bond of beneficial fellowship for a wider dissemination of scientific knowledge, in not only pulmonary diseases, but for all the kindred specialties dealing with the anatomy, physiology, and pathology of the chest. We rejoice with you in the progress which the College has made.

As their Chief Medical Director, I wish to express for the physicians in the Department of Medicine and Surgery of the Veterans Administration, deep appreciation to you for the privilege of joining, as searchers of knowledge with you, in the programs of your scientific sessions from year to year, and the favor of ad-

mission to the pages of almost every number of your official publication "Diseases of the Chest," whose editorial board has been most liberal in accepting scientific articles by physicians in the Veterans Administration, and very generous in its laudatory editorial comment at appropriate occasions on the techniques in clinical medicine and research, and the methods for control of tuberculosis practiced in our Veterans Administration hospitals and clinics. It has given us a sense of belonging and a satisfaction in contributing in a measure to the sum total of knowledge in diseases of the chest.

For myself, I am exalted by my selection as an honorary Fellow in the American College of Chest Physicians and it is a privilege to accept the diploma so designating me—not only as an honor for me but in recognition of the professional contributions of all the physicians in the Veterans Administration engaged in the art and practice of diseases of the chest. I thank you for this great honor.

Report of the Committee on College Essay Award, 1952-53

Dr. A. Link Koven, Philadelphia, winner of the 1953 College essay contest, receiving certificate and $250.00 award from Dr. Hugh L. Houston, Murray, Kentucky, at the President's Banquet, Hotel New Yorker, Saturday, May 30, 1953.

The Committee on College Essay Award, comprised of Drs. Hugh L. Houston, Murray, Kentucky, Chairman; E. R. Fenton, Washington, D. C.; Joseph S. Hiatt, McCain, North Carolina; Foster Murray, Brooklyn, New York; and David Salkin, San Fernando, California, selected the following as winners of the 1952 Essay Contest:

FIRST PRIZE *"Bronchogenic Carcinoma in Chromate Workers,"*
A. Link Koven, M.D., Philadelphia, Pennsylvania.

SECOND PRIZE *"Contra-Lateral Spontaneous Pneumothorax as a Complication of Intrathoracic Operations,"*
Adrian M. Sabety, M.D., East Orange, New Jersey.

THIRD PRIZE *"Endomyocardial Fibroelastosis,"*
William R. Halliday, M.D., Salt Lake City, Utah.

Dr. Hugh L. Houston, Chairman, presented the First Prize Certificate and $250.00 Award to Dr. Koven at the President's Banquet held at the Hotel New Yorker, New York City, May 30, at the 19th Annual Meeting of the College. Dr. Sabety was also present at the banquet to receive his Second Prize Certificate. The Third Prize Certificate of Honorable Mention was awarded in absentia to Dr. Halliday.

1954 COLLEGE ESSAY AWARD

The Committee, at its meeting on May 29, agreed upon the following rules for next year's contest:

The contest will be open to any medical student studying for the degree of Doctor of Medicine, on any phase relating to the diagnosis and treatment of heart and lung disease, and must be an original work.

The first prize will consist of a cash award of $250.00 and a certificate. The second and third prizes will be certificates of merit. The Essay Award is open to all medical students in accredited medical schools throughout the world.

The winning contributions will be selected by a board of impartial judges and will be announced at the 20th Annual Meeting of the American College of Chest Physicians, to be held in San Francisco, California, June 17-20, 1954. All manuscripts will become the property of the American College of Chest Physicians and will be referred to the Editorial Board of the College journal for consideration. The College reserves the right to invite the winner of the first prize to present his contribution at the Annual Meeting.

Applicants should study the format of *Diseases of the Chest* as to length, form, and arrangement of illustrations to guide them in the preparation of the manuscript. The following conditions must be observed:

1) Five copies of the manuscript typewritten in English (double spaced) should be submitted to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, not later than March 15, 1954.

2) The only means of identification of the author shall be a motto or other device on the title page and a sealed envelope bearing the same motto on the outside enclosing the name and address of the author..

3) A letter from the Dean or Chairman of the Department of Medicine of the medical school certifying that the author is a medical student studying for the degree of Doctor of Medicine and that the contents represent original work.

20th ANNUAL MEETING

The 20th Annual Meeting of the American College of Chest Physicians will be held in San Francisco, June 17-20, 1954. Physicians interested in presenting scientific papers on any phase in the diagnosis and treatment of heart and lung disease should send a 100-word abstract, no later than January 1, 1954, to Dr. Edgar Mayer, Chairman of the Committee on Scientific Program, 850 Fifth Avenue, New York 21, New York.

College Chapter News

ROCKY MOUNTAIN CHAPTER

The Rocky Mountain Chapter, encompassing the states of Colorado, New Mexico, Utah and Wyoming will present the following program on October 3 at the Shirley Savoy Hotel in Denver:

9:30 A. M.—

"Treatment of Tuberculosis with Isoniazid and with Isoniazid and Streptomycin,"
William F. Russell, Denver, Colorado.

"Carcinoma of the Lung Occurring in the Tuberculous Individual,"
William B. Condon, Theodore K. Gleichman and Robert P. Harvey, Englewood, Colorado.

"Preliminary Report on the Results of Treatment of Tuberculosis with Anti-tuberculosis Drugs,"
H. M. Van Der Schouw and Jerome D. Textor, Wheat Ridge, Colorado.

"Recent Trends in the Surgical Treatment of Bronchopulmonary Disorders,"
William F. Stone, Jr., Colorado Springs, Colorado.

12:00 — *Luncheon.*

Address: "Use of Digitalis and Quinidine,"
Colonel Edwin Goyette and Abe Ravin, Denver, Colorado.

2:00 P. M.—

"Cor Pulmonale,"
Colonel Edwin Goyette, Denver, Colorado.

"Management of Congestive Heart Failure,"
Abe Ravin, Denver, Colorado.

WISCONSIN CHAPTER

The following program will be presented by the Wisconsin Chapter on Sunday, October 4 at the Hotel Schroeder, Milwaukee:

1:00 P. M.— *Registration and viewing of exhibits.*

2:00 P. M.— *Scientific Program.*

"Electrocardiographic Problems in Daily Practice,"
George A. Hellmuth, Chicago, Illinois.

"Granulomatous Lung Disease,"
Joseph M. Lubitz, Milwaukee, Wisconsin.

"Surgical Treatment of Mitral and Aortic Stenosis
Alfred Goldman, Los Angeles, California.

"Inhalation of Dyhydrostreptomycin Dust in the Treatment of Diseases of the Respiratory Tract,"
Mary Karp, Edward E. Avery, et al., Chicago, Illinois.

"Diseases of the Trachea" (illustrated by motion picture),
Kenneth C. Johnston, Chicago, Illinois.

6:00 P. M.— *Dinner.*

Address: "Palliative Treatment of Inoperable Bronchogenic Carcinoma,"
Edgar Mayer, New York, New York.

PENNSYLVANIA CHAPTER

The Pennsylvania Chapter of the College, in conjunction with the American Academy of General Practice will hold a postgraduate course in Diseases of the Chest. This course will be held at the Pittsburger Hotel on four successive Sundays, beginning Sunday, September 13, 1953, and will be from 9:30 to 1:30.

On October 1, the Pennsylvania Chapter will meet jointly with the staff of the Cresson Sanatorium in Cresson, Pennsylvania. The chapter will hold a business meeting to be followed by a scientific program.

KENTUCKY CHAPTER

The Kentucky Chapter will meet in conjunction with the Kentucky State Medical Association in Louisville, on September 23. Dr. Paul H. Holinger, F.C.C.P., Chicago, Illinois, will be guest speaker at the chapter meeting.

SOUTHERN CHAPTER

An interesting program is being prepared by the Southern Chapter of the College, comprised of the 16 southern states and the District of Columbia, to be presented in Atlanta, Georgia, October 26-27, in conjunction with the meeting of the Southern Medical Association in Atlanta, October 26-29, 1953.

QUEBEC CHAPTER

A joint meeting of the Quebec Chapter of the College and La Societe de Phtisiologie de Montreal will take place on September 26 at l'Hopital Notre-Dame of Montreal at 10:00 A.M. The scientific program will consist of a symposium on pulmonary tumors. Guest speakers will be Richard H. Overholt, M.D., F.C.C.P., Clinical Professor of Surgery, Tufts Medical College, Boston, and Jules Prevost, M.D., F.C.C.P., Director of Clinical Instruction at l'Universite de Montreal and l'Hopital Notre-Dame, and Director of the Chest Disease Clinic. The discussion will be conducted in French and English by Gaetan Jarry, M.D. F.C.C.P., Medical Director of l'Institute Bruchesi, Inc. of Montreal and Edouard Gagnon, M.D., F.C.C.P., surgeon at l'Hopital Notre-Dame.

Book Review

THE TRANSACTIONS OF THE 12th CONFERENCE ON THE CHEMOTHERAPY OF TUBERCULOSIS, conducted under the auspices of the Veterans Administration, Army and Navy, Atlanta, Georgia, February 9th to 12th, 1953.

These transactions are recommended to all physicians, surgeons and laboratory investigators interested in the treatment of tuberculosis. The cooperative clinical investigations and research projects in the chemotherapy of tuberculosis, carried on by the many Veteran Administration, Army, Navy and civilian hospitals, have advanced our knowledge in this field tremendously. Fifty-six papers presented to the conference have been included in these Transactions. They cover a variety of subjects and bring the physician remarkably up to date on the latest advances in the treatment of tuberculosis. Many of the papers deal with isoniazid in the treatment of tuberculosis, including different dosages and durations, alone and in various combinations with streptomycin and/or PAS. Cooperative studies on various chemotherapy regimens under investigation are analyzed and the results summarized. Several papers on the treatment of non-pulmonary tuberculosis, particularly miliary and meningeal tuberculosis are included.

There are more than a dozen papers dealing with the surgical and laboratory studies of pulmonary tuberculosis. The significance of isoniazid-resistance is considered in some detail. Studies dealing with results of other antimicrobial agents such as Terramycin, Viomycin and Pyrazinamide in tuberculosis are included, as well as results in treatment of certain fungus diseases.

One of the high points in the Transactions is an admirable paper by Dr. William Tucker regarding the part that the Veterans Administration, Army and Navy hospitals have played in the development of our present knowledge of antimicrobial therapy of tuberculosis and the need for continuing such cooperative studies.

KARL H. PFUETZE, M.D.

Obituaries

LOREN LESLIE COLLINS

1890 - 1953

Few physicians have manifested the courage to discard theory, speculation and opinion, and fight tuberculosis with "bare fists" as did Loren Collins. He wasted no time on controversial matters as he was convinced that the disease can be eradicated by a few well established fundamental measures. Therefore, he struck straight at the heart of this great scourge, namely the tubercle bacillus.

He was born at Wilkes-Barre, Pennsylvania on October 24, 1890, and graduated from the Bloomsburg, Pennsylvania State Normal School in 1911. In 1917 he was granted the degree Bachelor of Physical Education from the American College of Physical Education, Chicago. He received the degree of Bachelor of Science from the University of Illinois in 1922. He then entered the medical school and graduated from the University of Illinois with the degree of Doctor of Medicine in 1925. The year 1924-1925 was spent on an internship at St. Vincent De Paul Hospital, Norfolk, Virginia. From 1925 to 1927 he was resident in pediatrics at the Minneapolis General Hospital and the next year he was resident in pediatrics at the University of Illinois Research Hospital.

From 1928 to 1938 Dr. Collins was staff physician at the Chicago Municipal Tuberculosis Sanitarium. There his diagnostic and therapeutic work, as well as investigative activities, were outstanding. This decade in close association with such physcians as Henry Sweany, Richard Davison and George Turner qualified him for most responsible positions. In 1938 he became medical director of La Salle County Tuberculosis Sanatorium at Ottawa, Illinois. The same year he was made medical director of the De Kalb County Sanatorium. He lived at Ottawa but served both of these institutions. In 1943 the Woodford County Sanatorium Commission convinced him that he should operate that institution. Thus he was medical director of three county sanatoriums but retained his home in Ottawa. During this period Dr. Collins became famous for his methods of combating tuberculosis. He was firmly convinced that every physician in each of the three counties he served should actively participate in the tuberculosis eradication program. Some of these physicians had never administered a tuberculin test. They had no x-ray facilities. He not only invited, but urged that they attend and assist in the administration of tuberculin tests in the schools. Soon all physicians in the three counties were administering this test routinely to persons of all ages in their offices. Those who did not have x-ray facilities were invited to send their tuberculin reactors to the sanatorium where Dr. Collins made, read, and reported findings without charge to the physicians as he was so anxious to reach every one and to find all clinical cases of tuberculosis as early as possible.

When clinical tuberculosis was found and the patients were admitted to the sanatorium, Dr. Collins kept their physicians in close contact with them. When they were ready for discharge, he always referred them back to their family physicians. Thus every doctor's office became a tuberculosis center. The physicians

of each county were interested in diagnosing tuberculosis in all stages of its evolution. They became informed about modern treatment and post sanatorium care. With such organization and activity tuberculosis was diagnosed so early that contagious cases became rare, thus tubercle bacilli were kept so controlled that the disease rapidly decreased.

By 1946 physicians of these three counties had become so expert in handling their local tuberculosis problems and the disease was so well controlled that Dr. Collins felt he could be of greater service in an area where tuberculosis was still prevalent. Therefore, he accepted the medical directorship of Madison County Sanatorium at Edwardsville, Illinois. There he soon had the physicians of the county participating in the program with the same excellent results as had been obtained at his previous location. Thus he demonstrated clearly in four counties that the medical profession, including specialists and general practitioners, can be actively enlisted in tuberculosis work, and that such organization is the surest and quickest way of eradicating the disease.

He was ever ready to accept any fundamental procedure which would increase the activity of the citizenry in the solution of its tuberculosis problem. He had adopted the program of the Committee on Tuberculosis of the American School Health Association for the certification of schools with reference to tuberculosis control work in progress. This had proved so successful elsewhere that he was determined to have every school in Madison County certified. This activity was well under way and 20 schools received certificates by the spring of 1953.

There was no physician in America who had a clearer vision of the tuberculosis eradication goal and no one who was traveling faster toward that destination.

Dr. Collins was always active in medical and tuberculosis organizations. He was a member of the National Tuberculosis Association and the American Trudeau Society. The Illinois Trudeau Society was organized in 1942 and he was elected secretary-treasurer and continued in that capacity until 1948. He was vice-president in 1949 and president in 1950. He served as president of the Mississippi Valley Trudeau Society in 1944-1945. Dr. Collins held Fellowship in the American College of Chest Physicians and had for sometime served efficiently as chairman of the Committee on Hospital Statistics.

He was an effective speaker and writer. His presentations were concise and never diluted with theory, speculation and personal opinion. He possessed so many facts, some of which he himself had established, that he had no time to waste on tangenital excursions. He firmly believed that the general practitioner is the cornerstone in tuberculosis eradication and a number of his published articles were directed to him. For example, "Advantages of Tuberculin Testing Surveys" (1942), "The Tuberculin Test in Tuberculosis Control" (1943), "Tuberculosis Control Depends Upon the Practicing Physician" (1946), and "The Part of Local Practicing Physicians in Tuberculosis Control in Schools" (1951).

Dr. Collins was a mild mannered, genial physician. Yet he possessed such courage of his convictions that he would admirably and successfully stand his ground against any attack on the facts upon which his program was established.

While attending the annual meeting of the American College of Chest Physicians in Chicago in May, 1944, Dr. Collins suffered a severe attack from coronary disease and was hospitalized for several weeks. However, he gradually resumed his activities. Although after partially recovering from the initial attack, for the remainder of his life he endured considerable pain and dyspnea, yet he worked to the limit of his capacity and beyond with his eyes fixed upon the tuberculosis eradication goal. While he was planning to attend the annual meeting of the American College of Chest Physicians held in New York City in May, he developed a severe coronary attack in the early morning of March 9, 1953 and was immediately admitted to the Passavant Memorial Hospital in Chicago. On two occasions I talked with him in his room by long distance telephone. Each time he said little about his health but devoted the conversation to his work, finally expressing the

hope that he might survive long enough to complete the manuscript which he was preparing on the importance of the general practitioner in tuberculosis. Although he showed evidence of improvement for a time he suddenly died on May 1, 1953. His death was a serious personal loss to me as it was to a host of other friends. For a quarter of a century I had true admiration for Dr. Collins and his work, and on our numerous occasions together I never failed to gain important information from him.

His enthusiasm, sincerity, accomplishments, vast store of knowledge, kindly manner and untiring efforts to eradicate tuberculosis, together with numerous other excellent qualities made Loren Collins one of America's truly great physicians. To emulate him and his work insure success to any individual or community.

J. ARTHUR MYERS

BEN E. GOODRICH
1900 - 1953

Dr. Ben E. Goodrich died suddenly of coronary artery occlusion on June 15. He graduated from the State University of Iowa in 1926, receiving degrees of B.Sc. and M.D. He served at Henry Ford Hospital, Detroit, Michigan, for 26 years as intern, medical resident, associate in the Cardio-Respiratory Division, and for the past four years as Chief of the Clinic on Pulmonary Disease. Dr. Goodrich served his country during World War I as an infantryman and in World War II he served in the Medical Corps of the Navy, emerging as a Captain.

Dr. Goodrich was the author of many scientific papers. He was a Fellow of the American College of Chest Physicians and served as President of the Michigan Chapter of the College in 1951-1952. He was also a Fellow of the American Medical Association and the American College of Physicians, and held membership in many other medical societies.

Dr. Goodrich is survived by his wife Martha; a son Dr. Frank Goodrich on the staff at Henry Ford Hospital; two grandchildren; his father, George W. of Gregory, Michigan; a brother John of Washington, D. C.; and three sisters, Mrs. Margaret Jensen of Harlan, Iowa, Mrs. Beth Hills of Birmingham, Michigan, and Mrs. Amelia Worden of Ypsilanti, Michigan.

WILLARD B. HOWES, M.D., Governor for Michigan

MAXIMILIAN POLLAK
1891 - 1952

On November 20, 1952, Maximilian Pollak died of coronary heart disease following a short period of illness in Memphis, Tennessee. He was born January 20, 1891 in Budapest, Hungary. He was graduated in Medicine from the Royal Hungarian University of Science in 1914. He entered the medical corps of the Austrian-Hungarian Army and was discharged as Colonel at the end of World War I. He then entered private practice in Budapest specializing in diseases of the chest.

Maximilian Pollak was a dynamic, energetic man. The fact that he had twice succumbed and took the cure for tuberculosis and that he was afflicted with severe arthritis did not diminish his enthusiasm or capacity for taking responsibilities. His booming voice added zest and flavor to discussion in many meetings. He had prophetic vision and applied considerable foresight and philosophy to his ideas. His principles were of the highest and he maintained them even at the cost of personal sacrifice. His death is a severe loss to the cause of clinical medicine and tuberculosis control.

OTTO L. BETTAG, Regent for Illinois

Modern Medical Monographs

CHRONIC PULMONARY EMPHYSEMA, PHYSIOPATHOLOGY AND TREATMENT. By Maurice S. Segal, M.D. and M. J. Dulfano, M.D. Copyright 1953 by Grune and Stratton, Inc.

Not many years ago, emphysema was considered in the group of chronic diseases for which no treatment was available. Not very much was known about it and the few therapeutic measures that were used seemed to give, at the best, mild and temporary palliation. In recent years, more attention has been directed to this disease; and a great deal of work has been done on etiological factors, and in the development of the successful methods of treatment. It is true that much remains to be done. But, the understanding of the pathology and the development of therapy has progressed to a point that all physicians, who are involved in chest disturbances of any sort, must be aware of this developing therapy. Under these circumstances, it is timely to have a monograph on chronic pulmonary emphysema.

This volume is particularly directed to those who have some special knowledge on both bronchial and pulmonary diseases. But, it is so written that those whose major interest is simply in learning how to handle a particular case of emphysema can also use it as a reference volume.

Quite properly, treatment of this disease is not discussed until the seventh chapter. The preliminary material dealing with development and pathology, function tests, and the clinical picture including complications. It is not necessary to emphasize the importance of all this material in its relation to treatment; that is well understood. It seems necessary to point out that the understanding of emphysema, particularly its development, pathology, and physiology, will make possible treatment at a much earlier stage and perhaps prevent the development of the far advanced case which is so troublesome both to the patient and to the physician.

In regard to therapy itself, the authors have emphasized, and I believe correctly so, that there are many factors in the course of treatment which may vary with the individual and with the case. They range from various drugs to inhalation therapy, with and without mechanical aids, and includes such procedures as pneumoperitoneum and breathing exercises. It is a large subject, and a difficult one to cover in a monograph. The result is a meaty book whose pages are filled with concentrated information.

Those who see only an occasional case of emphysema should read it carefully, because it gives a broad base upon which treatment can be developed. Those whose practice includes many emphysematous patients would do well to use this little monograph as a reference volume, since these patients notoriously show the most distressing individual variations and frequently do not continue to improve as much as is anticipated. For those who deal in industrial matters, who must consider degrees of disability, the chapter on pulmonary function tests and the appendix on methodology will be of additional value.

It is a well organized and concise volume, and should become a popular handbook.

EDWIN R. LEVINE, M.D.

COLLEGE EVENTS

NATIONAL AND INTERNATIONAL MEETINGS

Interim Session, Semi-Annual Meeting, Board of Regents,
St. Louis, Missouri, November 29-30, 1953.

20th Annual Meeting, American College of Chest Physicians,
San Francisco, California, June 17-20, 1954.

Third International Congress on Diseases of the Chest
American College of Chest Physicians
Barcelona, Spain, Fall, 1954

Tenth Congress of the Union of Latin American Societies Against Tuberculosis,
Caracas, Venezuela, December 5-10, 1953.

POSTGRADUATE COURSES

8th Annual Postgraduate Course on Diseases of the Chest,
Hotel Knickerbocker, Chicago, Illinois, September 28 - October 2, 1953.

6th Annual Postgraduate Course on Diseases of the Chest,
Hotel New Yorker, New York City, November 2-6, 1953.

CHAPTER MEETINGS

Kentucky Chapter, September 23, Louisville.
Quebec Chapter, September 26, Hopital Notre-Dame, Montreal, Quebec.
Pennsylvania Chapter, Cresson Sanatorium, October 1, Cresson, Pennsylvania.
Rocky Mountain Chapter, October 3, Shirley Savoy Hotel, Denver, Colorado.
Wisconsin Chapter, October 4, Hotel Schroeder, Milwaukee, Wisconsin.
North Carolina Chapter, October 16, Oteen and Asheville.
Southern Chapter, October 26-27, Atlanta, Georgia.

MEDICAL SERVICE BUREAU

POSITIONS WANTED

A well trained thoracic surgeon and medical director desires a position in an active hospital in the East, preferably in the New York-Boston area. Would prefer a directorship entailing work of actual clinical nature as well as administrative. Experienced in all phases of collapse therapy, and resections. Also has wide experience in bronchoscopy. Licensed in states of New York, Ohio, and Michigan. Would consider full time position as thoracic surgeon. Please address all inquiries to Box 279B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Experienced administrator and chest specialist, F.C.C.P., desires superintendency or medical directorship of a tuberculosis hospital. Outstanding qualifications. Excellent references from chest specialists of national repute. Please address all inquiries to Box 280B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

POSITIONS AVAILABLE

Staff physician wanted, Nebraska Hospital for Tuberculous. Experience desirable, but not essential. Salary dependent upon experience and qualifications. Should be eligible for Nebraska license. Give details concerning qualifications and references. Please address all inquiries to Box 269A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Staff physicians wanted for a tuberculosis hospital located in Southern California. Salary depends upon qualifications. No tuberculosis experience necessary. Please address all inquiries to Box 273A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Wanted: assistant or associate medical director preferably under 50. New modern 200 bed tuberculosis hospital. Salary up to $10,000 depending upon experience. Alabama license necessary. Unusually complete laboratory, x-ray facilities. Medical Director 38 years old, F.C.C.P., Assoc. A.C.P., board certified Internal Medicine. Please address all inquiries to Medical Director, District No. I, Tuberculosis Hospital, Decatur, Alabama.

The Veterans Administration Hospital, Downey, Illinois, 35 miles from Chicago, has several vacancies for physicians in the 297-bed tuberculosis unit of the hospital. Salaries determined by experience and qualifications: Department of Medicine and Surgery, $5,000 to $9,600 per annum. Applicants must be between ages of 21 and 54, hold degree of Doctor of Medicine from approved institution, and must have completed approved internship, as well as posess license to practice in a state or territory of the U. S., and must meet physical requirements. Write to the Manager, Veterans Administration Hospital, Downey, Illinois.

Position open for tuberculosis specialist with government of Guam. Position offers unlimited opportunities for personal scientific advancement. Excellent opportunity for eager and enthusiastic professional man to make enviable record in the control of tuberculosis in this American Territory. Two year contract offered; transportation paid; salary $7,800 to $10,925. For further information, write to the Director of Labor and Personnel, Government of Guam, Agana, Guam, Marianas Islands.

ANNOUNCEMENTS

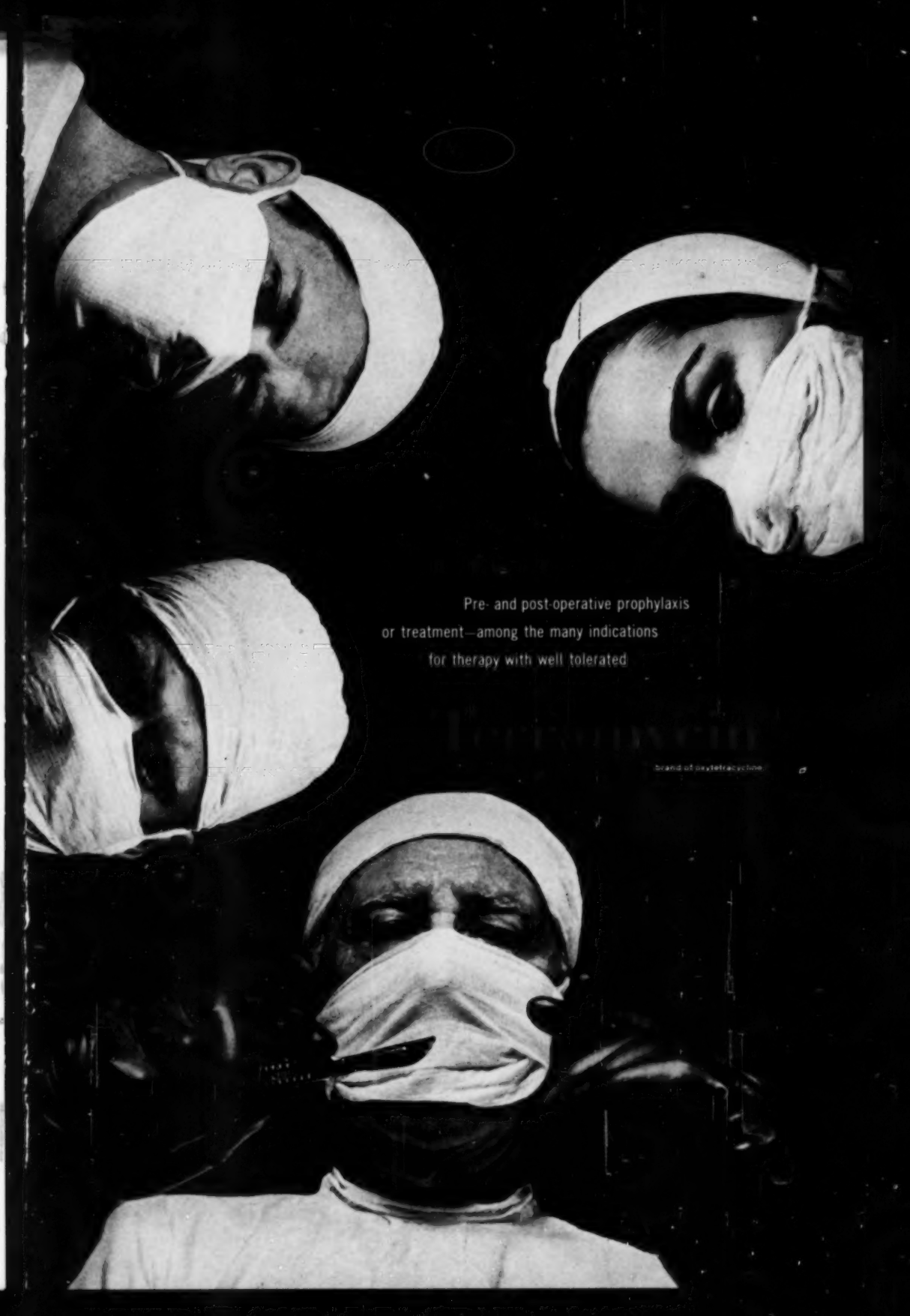
The Second International Congress of Cardiology will be held in Washington, D. C., September 12-15, 1953. Dr. Paul D. White, Honorary Fellow of the American College of Chest Physicians will serve as Chairman. It will be followed by the Annual Scientific Session of the American Heart Association, September 16-18, 1953. Physicians desiring additional information may write to the Director, L. W. Gorham, M.D., 44 East 23rd Street, New York 10, New York.

Under the auspices of the Minnesota Heart Association, the University of Minnesota will present a Symposium on Cardiovascular Physiology and Surgery, September 14-16, 1953 in the Museum of Natural History Auditorium on the campus. A host of internationally known physiologists and vascular surgeons will participate. The symposium will be open, without tuition fee, to all physicians and to qualified investigators in the field of cardiac physiology. Further information may be obtained from the Director, Department of Continuation Medical Education, University of Minnesota Hospitals, Minneapolis 14, Minnesota.

Dr. Raman Viswanathan, Regent of the College for India and Director General, Health Services of India, reports that the Chest Institute, Delhi University, Delhi, India, established for the purpose of postgraduate teaching in research of chest diseases, is in need of teaching equipment. He has requested teaching material such as back issues of medical journals, lantern slides, films, pathological specimens, books, and research equipment. Members of the College and other readers of the College journal who wish to contribute any of the abovementioned material are requested to communicate directly with Dr. Viswanathan, Director, Chest Institute, Delhi University, Delhi, India.

Under the direction of Professor Jose F. Verna, the Instituto y Catedra de Tisiologia of the Universidad Nacional de Cordoba (Argentina) will present the 7th Improvement Course in Phthisiology, Sept. 21 - Oct. 3, at the Hospital Transito Caceres de Allende.

The New York Tuberculosis and Health Association announces that the James Alexander Miller Fellowship for Research in Tuberculosis will be available from July 1, 1954 to June 30, 1955. Application forms will be supplied on request and must be submitted by October 1, 1953. Write to: New York Tuberculosis and Health Association, 386 Fourth Avenue, New York 16, New York. Awards will be announced December 1, 1953.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